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COMPLICATIONS OF THE SURGERY OF PATENT DUCTUS ARTERIOSUS

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THIS report concerns the complications of the surgery of patent ductus arteriosus in a series of sixty-one successive patients. Fifty-three operations were ligations in continuity, and eight were divisions employing the modification of the technique of Gross.[1] All of these patients had a presumably clinically uncomplicated ductus arteriosus, and none of them were believed to have had subacute bacterial endarteritis before operation. The diagnosis of patent ductus arteriosus was clear-cut and was confirmed upon operation in every case.

The youngest patient was just under 3 years and the oldest was 35 years of age. Thirty-three (55 per cent) of the patients were younger than 10 years, twenty-three (37 per cent) were between the ages of 10 and 20 years, and five (8 per cent) were older than 20 years (Table I). The eight patients in whom a division of the ductus was carried out were between the ages of 3 and 6½ years.

TABLE I. AGE INCIDENCE

Age	Under 5	5-9	10-14	15-19	20-29	30-35
No. cases	17 (28%)	16 (26.5%)	12 (19.5%)	11 (18%)	4 (6.5%)	1 (1.5%)

Virtually all of the patients developed some pleural effusion postoperatively, but in only twenty-two (36 per cent) was aspiration deemed necessary, three of whom were aspirated twice. In two of the twenty-two, only air (250 c.c. and 150 c.c.) was obtained. In the remaining twenty, serosanguineous fluid in varying amounts of between 50 and 450 c.c. was obtained by aspiration. Penicillin intrapleurally or intramuscularly, and the sulfonamides intrapleurally or orally were not used, either pre- or postoperatively, except in patients developing a

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complication indicating their specific need, and in none of this series were these drugs employed as prophylactic treatment.

Postoperative pulmonary atelectasis occurred in five patients (8 per cent), all children, each time in the left lower lobe. Their lungs had been inflated at intervals routinely during the surgery and all atelectatic areas cleared before closure of the chest, the pleural cavities had been aspirated after closure, and inhalations of carbon dioxide had been administered for several days following surgery. All five patients recovered following frequent postural treatment and more vigorous and frequent inhalations of carbon dioxide, and in three cases small doses of sulfonamides were given orally. Penicillin was not available at the time.

Left recurrent laryngeal nerve paralysis occurred three times in this series. In two patients it has persisted, and in one total recovery was noted within a few weeks after operation. In spite of having identified the nerve in every instance at the beginning of the dissection of the ductus and having been careful not to injure it during the operation, this complication occurred. All three were in cases in which the patent ductus arteriosus was ligated and it is very likely that the pressure of the tapes and their knots and the scar tissue about them subsequent to the edema immediately following surgery accounted for the permanent paralysis, for the nerve was intact after the ligation was completed. In the two patients (both adult females) with persistent paralysis, the quality of the voice improved considerably over a period of several months after operation, but the appearance of the vocal cords had not changed.

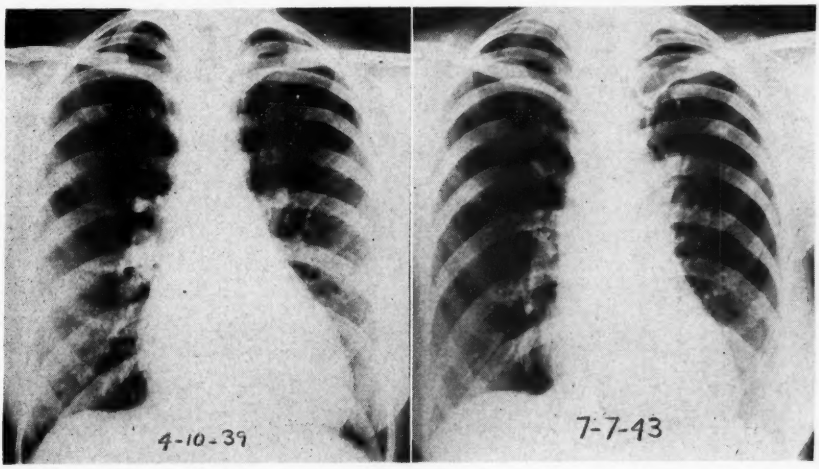
Two of the patients developed an empyema postoperatively. The first was a 16-year-old girl who presumably did not have a pneumonia, but developed a *Staphylococcus aureus* infection in the pleural space after the cultures of the first aspirated fluid were negative. She had a rib resection, and, while the lung was reluctant to re-expand, she finally made a complete recovery from the empyema. The other recently operated patient, a boy of 3 years, developed a pneumonia in the left lung following division of the ductus arteriosus. The lung consolidation and high fever came on thirty-six hours after operation, and he developed an empyema in spite of prompt chemotherapy (sulfadiazine and penicillin). Aspiration and instillation of penicillin into the pleural cavity was employed, but the fibrin clots finally demanded rib resection. He is making a good recovery but the tube is still in place. There was but the one pneumonia in this series.

There were no wound infections. The high incidence of keloid in the supramammary scars has forced us to abandon this approach through the second intercostal space anteriorly. In children as well as adults, if the anterior approach is preferred, the incision should be an inframammary one, for certainly in adult males and children the tension of the pectoralis muscles is the great factor in keloid formation rather than the weight of the breast. In the last twenty cases we have employed a posterolateral approach (resection of fourth or fifth ribs) and we believe it affords a far better exposure for any operation in the area of the ductus arteriosus. All our divisions have been carried out through this approach, and the incidence of keloid in the scars has been greatly diminished.

A serious distressing complication following ligation of the ductus arteriosus in our experience has manifested itself by sudden copious hemoptysis. Two case histories are briefly outlined with their follow-up.

CASE REPORTS

A. J., 35-year-old housewife, had an uneventful convalescence following ligation of the ductus arteriosus Feb. 22, 1943, and was discharged from the hospital thirteen days later. On July 3, 1943, after a strenuous day of housecleaning, she coughed up an ounce of bright red blood. The next night she awakened with rattling in her throat and had a similar experience with hemoptysis. The following two days she had an aching across and between the shoulders and raised more dark bloody mucus plugs before being admitted to the hospital July 7. She said she felt well otherwise. The temperature, pulse, and respirations were normal and the blood pressure was 120/90. Examination revealed a heart that was definitely smaller than before operation and now within the limits of normal and only a slight pulmonic systolic murmur could be heard. The blood counts were well within the normal range and electrocardiogram was not abnormal. The roentgenogram (Fig. 1, *A* and *B*) revealed a slight haziness in the left upper lung field and a slightly increased bulging of the left upper mediastinal border in the region of the pulmonary conus, with a normal contour of the heart.

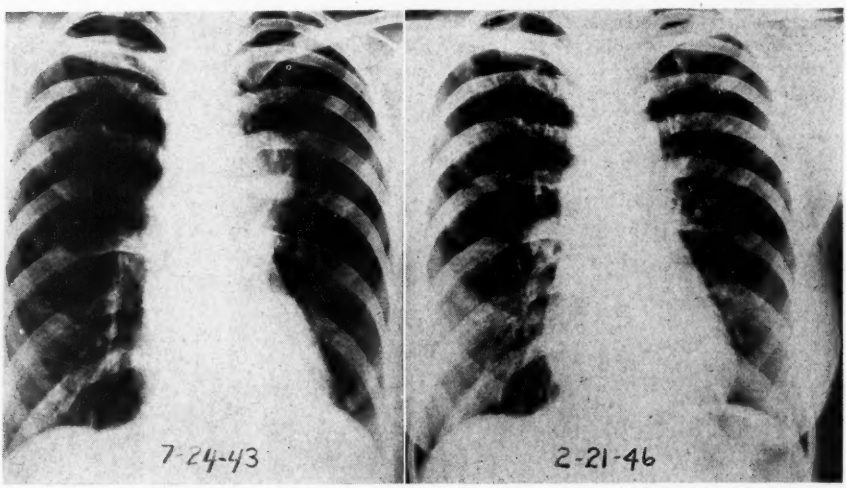


A. *B.*

Fig. 1.—*A*, roentgenogram of A. J. before operation showing prominence of the pulmonary conus and enlargement of heart. *B*, roentgenogram four days after onset of hemoptyses. Bulging in the area of the pulmonary conus and haziness of the mediastinal aspect of the left upper lobe is noted.

Roentgenogram twenty days after the onset of hemoptyses disclosed an increase in the mediastinal bulge and diffuse haziness in the medial aspect of the left upper lobe (Fig. 2, *A*). She remained in the hospital for six weeks with occasional small hemoptyses, almost daily streaking of mucus and some occasional aching pain between the spine and the left scapula above the fourth dorsal vertebra, but was discharged home in good condition to remain in bed. On Nov. 19, 1943 she had a sudden hemoptysis of 350 c.c. and was readmitted to the hospital. She was not in shock, the pulse was 100, blood pressure 130/80, and hemoglobin estimation was 86 per cent. Physical examination revealed râles over the left lung field and a persistent pulmonic systolic murmur, but no continuous or diastolic murmur. Roentgenogram and electrocardiogram remained the same. She had another hemoptysis of 200 c.c. of bright blood three days later, and was discharged eleven days after admission with no com-

plaints other than a little irritative cough and occasional streaking. A week later she had a massive sudden hemoptysis of 500 to 750 c.c. at home, and a transfusion was administered the following day because of a hemoglobin estimation of 63 per cent and 3,840,000 red blood cell count. On Jan. 8, 1944, another 500 c.c. hemoptysis occurred, then only streaking occasionally until February, 1945, when she had a 200 c.c. hemorrhage and none since then. She was last seen on May 17, 1946, and appeared well (Fig. 2, *B*). She volunteered that she had occasional streaking of her mucus in the morning, had led a normal active life with no limitation for over a year with no frank hemoptyses, and weighed 136 lbs. Pulse was 85, blood pressure 118/80. She had a low-grade systolic pulmonic murmur while sitting which disappeared in the supine. The lung fields were clear to auscultation. Fluoroscopy revealed the heart to be normal in size and contour, the lung fields were clear throughout; there was no conus fullness, and the previous bulging of the left upper mediastinum had disappeared. Electrocardiogram and blood counts were within the normal range.

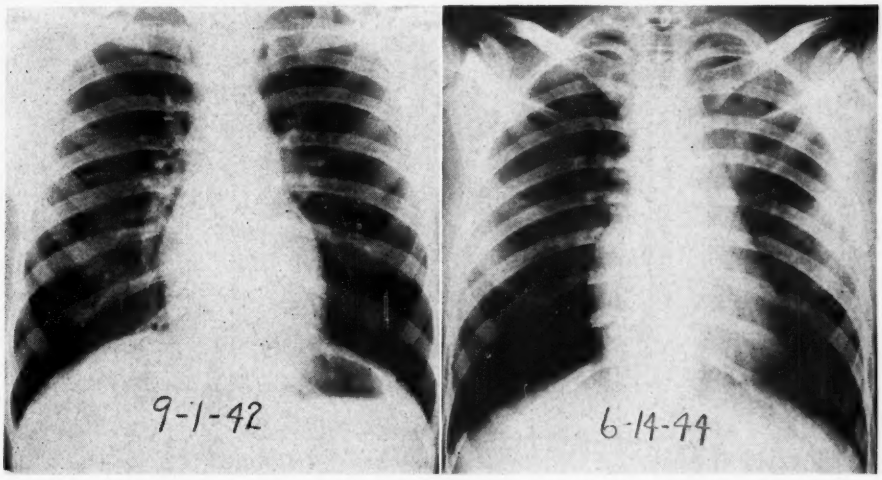


A. *B.*

Fig. 2.—*A*, roentgenogram of A. J. twenty-one days after initial hemoptysis showing increased mediastinal bulge and upper lobe opacity. *B*, roentgenogram one year after the last hemoptysis. Patient felt well.

J. S., female, 11½ years old, had her patent ductus arteriosus ligated in continuity with two silk umbilical tapes on Sept. 14, 1942 (Fig. 3, *A*). Convalescence was uneventful, there remained no cardiac murmurs, and she was discharged in excellent condition thirteen days later. She remained well without symptoms and without abnormal physical findings on examination until June 13, 1944 (twenty-one months after operation), when she coughed up a mouthful of bright red blood. She did not feel ill and had no other symptoms, but was admitted to the hospital for study. Temperature remained normal. The pulse was 100, and respirations 20. Blood pressure 140/100 on admission receded to 96/68 the following day. Moist râles were heard in the left lung, but cardiac findings and electrocardiogram were normal. X-ray of the chest (Fig. 3, *B*) on admission showed slight haziness of the left upper lobe which had cleared in forty-eight hours. Three hours after admission, while lying quietly in bed, she had a sudden hemoptysis of 4 oz. of bright blood. She was discharged after three days' observation. The following day, June 18, 1944, she had another 4 oz. hemoptysis and x-ray immediately afterward again showed only a slight haziness of the left upper lobe which cleared in forty-eight hours, and the physical findings were as before. Bronchoscopy June 22 was negative, as was the examination of the heart and lungs. She had six recurrences of the hemoptyses (Fig. 4, *A*) the last of which amounted to 500 c.c., and

she was admitted to the hospital Nov. 1, 1944, with temperature 100.2° F. (rectal), pulse 84, respirations 20, and blood pressure 94/56. She had a soft systolic murmur at the apex, but no other murmur. The left upper lung was slightly hazy by roentgenogram. The hemoglobin was 64 per cent, red blood cell count 3,470,000, white blood cell count 11,450. On November 2, the thorax was explored through a posterolateral approach (resection of the fifth rib) under endotracheal ether-oxygen anesthesia. The lung was adherent to the anterior chest wall at the site of approach for the ligation of the ductus arteriosus. A knuckle of lung, 2 cm. in diameter (mediastinal aspect of the left upper lobe) was densely adherent to the aorta at the site of ligation of the patent ductus arteriosus, but there was no thrill. It was as though the lung had been drawn into the hollow of the arch of the aorta, adjacent to which was an area of indurated firm lung the size of a walnut. While dissecting the lung from the aorta traversing recent and old organized blood clot, granulomatous appearing tissue exuded and two silk (umbilical tape) knots popped out (Fig. 4*B*). When completely freed from the aorta there was seen a 2 cm. cavity in the lung which connected with an open bronchus. Suddenly a forceful, 4 mm. stream of blood came from the aorta just beyond the site of the ductus ligation and from the granulomatous appearing area of the aorta.



A. *B.*

Fig. 3.—*A*, Roentgenogram of J. S. before operation. *B*, Roentgenogram a few hours after initial hemoptysis, showing bulging at the conus arteriosus and haziness of the left upper lobe.

This was immediately controlled by digital pressure. It was obvious that the two silk knots had eroded both the aorta and the lung and that a fistulous tract between the aorta and bronchus had been established. Five fine (00000) silk sutures in the wall of the aorta closed the opening and this was reinforced by suturing over it a layer of mediastinal pleura. The bronchial opening and the cavity in the lung were obliterated with fine catgut sutures on atraumatic needles. The lung was re-expanded, 2 Gm. of sulfanilamide crystals and 25,000 units of penicillin in 50 c.c. of saline were left in the pleural cavity, and the chest wall was closed without drainage. The blood pressure before operation was 90/60, at the close it was 90/70. During the latter part of the operation, 250 c.c. of blood was given. She had an uneventful convalescence and was discharged Nov. 7, 1944, fifteen days after operation (Fig. 4*C*). She has been well ever since and has no abnormal physical findings referable to heart or lungs (Fig. 4*D*).

We believe that both the above patients had identical lesions, an aortobronchial fistula, in spite of the fact that the first was not proved by operation.

In both there was no recurrence of the continuous murmur and no evidence of a fistulous tract from the pulmonary artery to the aorta, and the patency of the ductus has surely been obliterated. Both patients had similar clinical histories and roentgen findings. The magnitude of the hemoptyses in the first patient makes it unlikely that the silk knots eroded only the lung and that the bleeding could be from the lung itself. In spite of the fact that the patient has not had frank hemorrhage for fifteen months and the lesion appears to be healing, the fact remains that occasional streaking in the mornings occurs and there may be recurrences requiring surgical intervention.

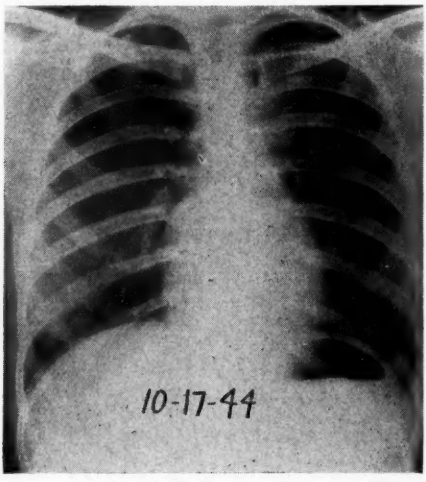


Fig. 44.—Roentgenogram of J. S. four months after onset of hemoptyses.

The mediastinal pleura over the site of the ligation of the ductus with two silk umbilical tapes tied side by side was not closed or sutured in either patient, and it may be that if it had been closed, this complication could have been avoided. The mediastinal pleura has been closed, burying the knots in all, since this experience. This procedure would protect the mediastinal aspect of the upper lobe but would not prevent erosion of the aorta or possible aneurysm. At any rate, this complication is a definite argument in favor of the division of the ductus arteriosus.

There were two deaths in this series. The first patient (J. D.) operated May 6, 1939, was discharged from the hospital fourteen days later following an uneventful recovery. She resumed normal activity and thirty days later developed a high fever and died of *Staph. aureus* endarteritis forty-eight days following ligation. At autopsy, a re-established fistula between the pulmonary artery and the aorta, around the ductus ligatures, and an aneurysm at the ductus were found. The ligatures had cut through the wall of the ductus arteriosus and a superimposed blood stream infection resulted in vegetations on both the pulmonary artery and aortic walls. The details of this case were presented at the Los Angeles meeting.[2]

The other death was an operative mortality. W. W., male, 6 years old, was diagnosed as having patent ductus arteriosus on his admission to the Children's Hospital in 1942 for

the treatment of pneumonia. Roentgenograms revealed bilateral pneumonia and a large, round heart consistent with a congenital lesion, and a fullness of the conus arteriosus. He made a rapid recovery and was observed for over three years. He was thin and underdeveloped and had not been able to carry on with others his own age. On admission to the hospital April 20, 1946, the pulse was 108, and blood pressure 92/38. Examination revealed a symmetrical chest that bulged forward, and a very active precordium. The lungs were clear. The point of maximum impulse was in the fifth interspace at the anterior axillary line, and there was a great continuous thrill over the pulmonic area. A loud, harsh, continuous machinery murmur over the pulmonic area was transmitted over the entire precordium and could be heard throughout the chest. His roentgenogram revealed a moderate cardiac enlargement with clear lung fields. Electrocardiogram before admission was not remarkable, and his blood counts were normal. On April 22, at operation, it was noted that the

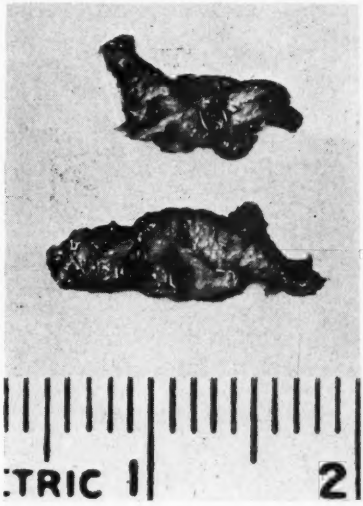


Fig. 4*B*.—Two silk umbilical ties and knots encased in fibrous tissue removed at operation. Patient J. S.

cardiac action was considerably more vigorous than was usual in these cases and the ductus arteriosus stood out more prominently and seemed stiffer, firmer, and thicker than the average ductus we had seen. Dissection around the ductus arteriosus was completed easily and while not being disturbed, the posterior pulmonary artery end of the ductus gave way with a resultant large hemorrhage. Two tapes passed around the ductus and tied failed to control the bleeding, but after forceps were applied, the bleeding stopped, the tapes were removed, and the ductus was divided. The aortic aspect of the ductus was sutured with fine silk and that forceps removed. One small leak in the aorta was closed with an interrupted suture. The pulmonary side of the ductus was then sutured distal to the forceps. Blood transfusion had been instituted at the onset of the hemorrhage. When the forceps was removed from the pulmonary side of the ductus, there was another, larger gush of blood proximal to the suture line which was fairly well controlled by digital compression while two interrupted mattress sutures were taken in the leaking pulmonary artery. This completed hemostasis, but at this point cardiac arrest intervened, and in spite of the usual restoration measures, we were unable to stimulate cardiac action. He had lost considerable blood at a rapid rate on two occasions, and we were unable to replenish the loss quickly enough to resuscitate the heart. The more pertinent findings at necropsy are outlined. The heart weighed 212 Gm. (normal, 94 Gm.) and was considerably enlarged. There were areas of pale, white, thickened epicardium over

the apex and right ventricle. Numerous small verrucose nodules were noted over the left auricular appendage, having the appearance of vegetations. The myocardium was increased in thickness, the papillary muscles hypertrophied, and the chordae tendinae were thicker and less transparent than usual. The endocardium was smooth and glistening, except for that over the mitral valve and immediately above it. Here the endocardium was thickened and not transparent, and was white. The mitral valve was increased in thickness, and had a nodular appearance and feel to it. The foramen ovale was 2 mm. in patency. The ductus arteriosus was absent at its two points of origin in the pulmonic and aortic aspects. It had apparently recently been closed and numerous fine sutures were in place. The inflammatory lesions of the heart, exclusive of the congenital lesions, were reported as consistent grossly and microscopically with rheumatic heart disease, although no typical Aschoff's bodies were found. Blood cultures were negative in ten days. The pathologist's diagnosis was cardiac hypertrophy and dilatation, chronic mitral endocarditis, pericarditis, and focal myocarditis.

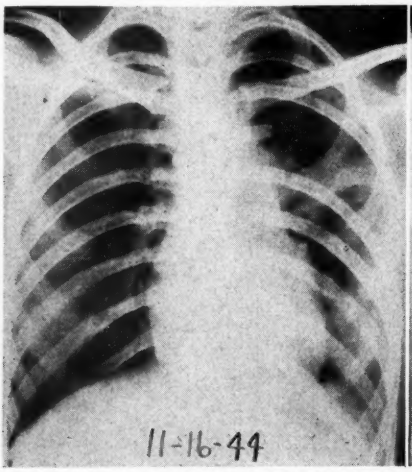


Fig. 4*C*.

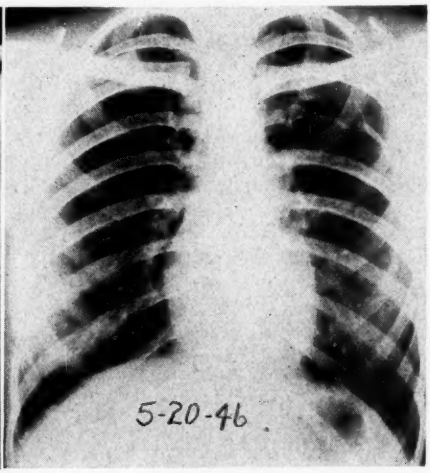


Fig. 4*D*.

Fig. 4*C*.—Roentgenogram of J. S. on discharge, two weeks after exploration and repair of aortobronchial fistula.

Fig. 4*D*.—Roentgenogram of J. S. eighteen months after discharge from hospital.

The ligation or division of the ductus arteriosus, had it been successful, might possibly have decreased the myocardial load and lengthened this patient's life, but we were ignorant of his having rheumatic heart disease at the time of his operation; we would not recommend surgery for patent ductus arteriosus complicated by rheumatic fever.

While this, the only operative death in the series, was a division of the ductus arteriosus rather than ligation, the death cannot be attributed to the division per se, for the end result would probably have been the same regardless of the method employed. The ligation with tape utterly failed to arrest the hemorrhage, but it was controlled by the division of the ductus arteriosus, and in retrospect, in this instance at least, it would have been judicious to have proceeded with division at once.

Probably the most embarrassing complication of the ligation of the patent ductus arteriosus from the standpoint of the surgeon is the persistence or recurrence of the continuous murmur. In fifty-three ligations, there were five patients (9.4 per cent) whose continuous murmur was noted within the first few

days after operation (as soon as the dressings were removed and an examination was permitted). The persistence of the murmur can be explained only by a failure to tie the tapes tightly enough to occlude the lumen of the ductus completely. Five others (9.4 per cent) were discharged from the hospital either with no murmur or a systolic pulmonic murmur, only to turn up later with a recurrence of the continuous murmur and usually some palpable thrill. In this group, the murmur was detected five or six weeks after ligation when the patient returned for examination, and presumably is explained by re-establishment of the fistula after the tapes had cut through the wall of the ductus arteriosus. We have not operated a second time on any of these ten cases. In some the murmur has remained unchanged, in others it has increased or diminished in its intensity; in seven of the patients, the murmur is now not as loud as before surgery, in two it is about the same as before ligation, and the remaining patient (J. D.) is dead.[2] In the seven cases surviving the division of the ductus arteriosus, there have been no recurrences of the murmurs nor any complications attributable to this method of treatment.

There are fifty-nine (96.5 per cent) patients alive, nine of whom have a persistent continuous murmur after the ligation of the ductus arteriosus. All of these patients are well with one exception, an adult female with an early recurrence of the murmur, a persistent vocal cord paralysis, diabetes, and a low reserve.

SUMMARY

1. Sixty-one cases of patent ductus arteriosus have been operated on; fifty-three (87 per cent) were ligated in continuity, and eight (13 per cent) were divided and sutured.

2. There were two deaths, one a late death due to *Staph. aureus* endocarditis, and one operative death (exsanguination and cardiac arrest).

3. Two patients developed an aortobronchial fistula with large hemoptyses, one of which was repaired surgically; the other appears to be healing spontaneously.

4. Five of the ligations had an early recurrence of the murmur due to incomplete occlusion of the vessel by the tapes, and five others had a late establishment of the fistula due to the tapes partially cutting through the wall of the ductus.

5. Five had left lower lobe postoperative atelectasis.

6. There were two empyemas, one of which followed a pneumonia.

7. Two patients have a permanent, left, recurrent, laryngeal nerve paralysis.

8. Fifty-nine patients are alive, and, in spite of nine patients having a persistent continuous murmur, only one is not well. She is a diabetic with a re-established fistula and a low reserve.

REFERENCES

1. Gross, R. E.: Complete Surgical Division of Patent Ductus Arteriosus, Surg., Gynec. & Obst. **78**: 36-43, 1944.
2. Jones, J. C., Dolley, F. S., and Bullock, L. T.: The Diagnosis and Surgical Therapy of Patent Ductus Arteriosus, J. THORACIC SURG. **9**: 413-430, 1940.

COMPLETE DIVISION FOR THE PATENT DUCTUS ARTERIOSUS

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DURING the last eight years, studies from this and other clinics have amply demonstrated that surgical therapy is an important method for the treatment of individuals with a patent ductus arteriosus. In the early stages of these surgical endeavors, closure of the ductus was always attempted by some form of ligation. It was fully realized that ligation of a vessel "in continuity" had certain limitations, because this procedure might not give complete closure of the shunt, or else there might be partial recurrence of the fistula if the ligatures cut through the vessel wall. While it was obvious that complete division of the ductus would be desirable, limited operative experience at that time made me feel that this was too hazardous an undertaking. Therefore, ligation alone was done, and in the great majority of cases this gave a good result and could be accomplished with a relatively low risk.

In the first part of the work, forty-three individuals of varying ages and with manifold complications of a persistent ductus were operated upon by some form of ligation. In most of these, two ligatures of No. 8 braided silk were used. In a few instances, narrow umbilical linen tape was employed. In twenty-nine cases, double ligatures were placed and then Cellophane was wrapped over these, the author believing that subsequent fibrosis excited by this material would cause some regional scarring and close off any small shunt which might have been left at the end of operation. The addition of a cellophane wrapping was an improvement, but it could not be relied upon to give a permanent closure of the shunt in all cases. A careful follow-up study on these forty-three patients (forty of whom survived surgical treatment) shows that in general, about 80 per cent obtained complete closure of the ductus and that the vessel remained closed. In about 10 per cent of the cases there was apparent complete closure of the ductus at the end of the operative procedure, and for a week or ten days thereafter, but subsequently the ligatures partially cut through the ductus and some of the fistula was re-established. In the remaining 10 per cent of the cases, the ligatures were not tied quite tightly enough, so that a small shunt was left at the close of the operation. In these last two groups the results were somewhat disappointing, but this does not vitiate the fact that most of these individuals were greatly improved because the work of the heart was tremendously reduced, and in some cases a *Streptococcus viridans* infection was permanently cured. While this general therapeutic picture might be classed as quite satisfactory, an attempt has now been made to develop a technique for complete division of the ductus in

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all cases. This goal appeared difficult to attain, but the search was stimulated by the conviction that division of the ductus would be the only operation which could give assurance of a complete and a permanent closure of the shunt.

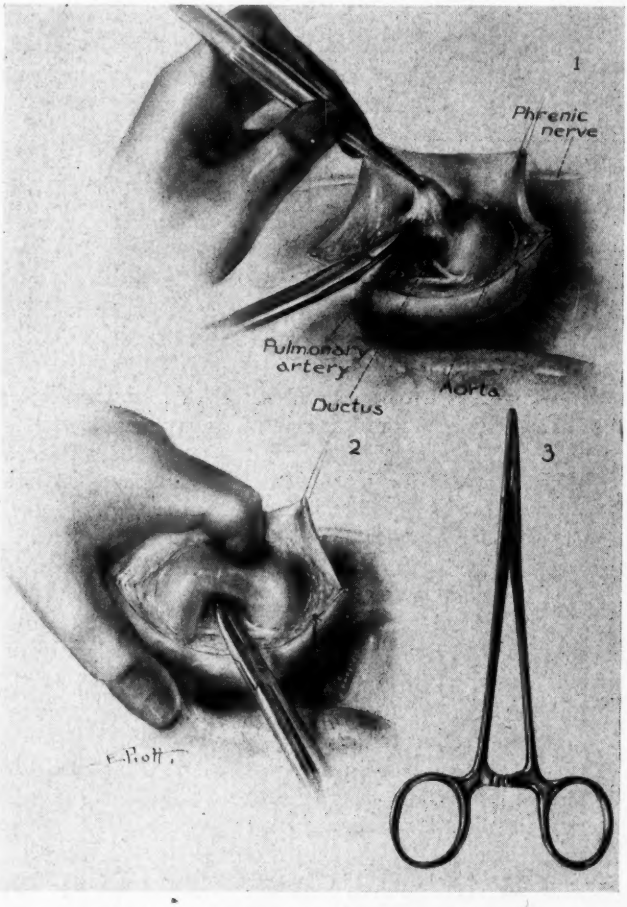
As far as I am aware, Touroff[5] was the first (Jan., 1940) to divide completely a patent ductus arteriosus. In 1941, I divided the ductus in a child of 10 years, and in 1944 reported the technique[4] which had been successfully used in fourteen cases. To date, I have divided the ductus in 90 patients, and would like to indicate briefly the technique which is currently employed. This is essentially the same type of operation, with but minor changes, as that which was described in the publication of 1944.

OPERATIVE TECHNIQUE

A route for exposure of the superior mediastinum and great vessels has been rather fully considered in other communications.[2, 3] Only a few points will be stressed here regarding this approach. The patient is placed on the table with a sandbag underneath the left side of the chest and left shoulder. The left arm is extended upward along the head. A long, curved incision is made beneath the left breast extending well around to the posterior axillary line. The entire breast is dissected free and raised from the pectoral fascia so that the breast can be turned upward, a point of extreme importance in gaining an adequate exposure in adult females. The pectoral muscles are divided just above their attachment to the thoracic cage. The anterior border of the latissimus dorsi muscle must be severed for an inch or two. The anterior serratus muscles are cut as far back as the long thoracic nerve. The left pleural cavity is entered through the third intercostal space, the intercostal muscles being divided from the sternal border around almost to the angle of the ribs. The third and second costal cartilages are cut so that the ribs can be pushed upward and held in that position with self-retaining retractors. It is extremely important to obtain a maximum opening from these various steps, in order to have sufficient room when working inside the chest.

The superior mediastinum is entered by a long cephalocaudal incision of the parietal pleura, this cut being made about one-half inch behind the phrenic nerve. The highest intercostal vein which courses over the mediastinal structures to reach the left innominate vein must usually be divided. The vagus and left recurrent laryngeal nerves are carefully searched for and spared from injury. Lymph nodes which obscure the field must be dissected away before exposing the underlying structures. Fat and areolar tissue must be cleared away from the terminal portions of the aortic arch and the near-by pulmonary artery so that the intervening ductus can be clearly visualized. In most subjects, a small lappet of pericardium extends upward and partially overlays the left lateral surface of the ductus. This must be completely removed from the ductus (so that it will not be subsequently included in the clamps which are placed upon the ductus). By sharp and blunt dissection (Fig. 1) this lappet can be pushed away. It is well to complete this step without perforating the pericardial sac; this precaution prevents pericardial fluid from running into the field.

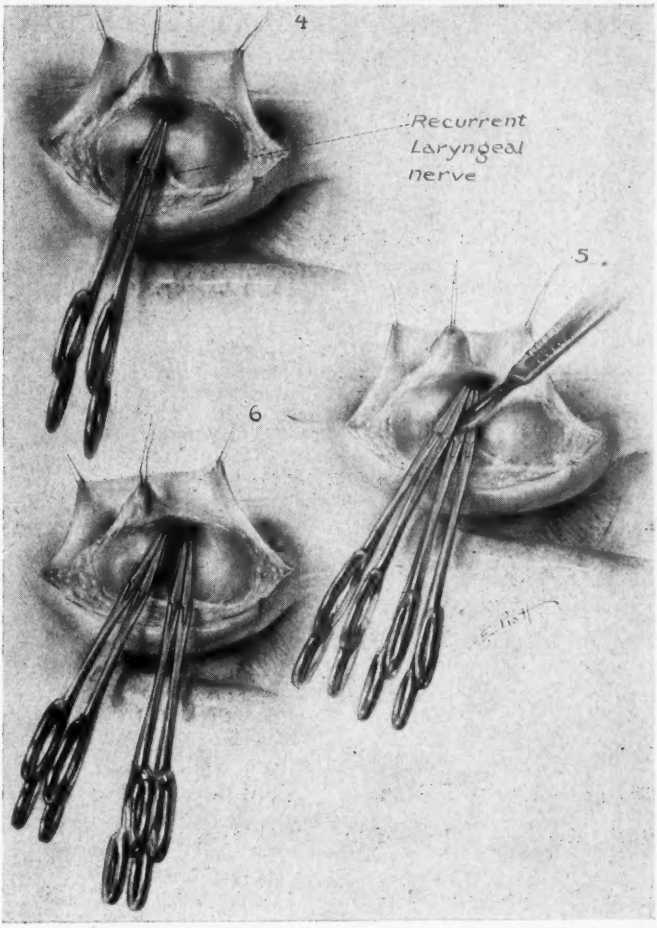
The posteromedial wall of the ductus is now circumvented by blunt dissection, which is most easily done with a right-angle clamp (Fig. 2), guiding the instrument by feeling the point with the tip of the left index finger. In this way, the right-angle clamp is carefully pushed between the wall of the ductus



Figs. 1, 2, 3.—*1*, Lappet of pericardium being turned caudally by dissecting it off of the ductus arteriosus. *2*, Back (medial surface) of ductus being freed by blunt dissection. *3*, Type of instrument to be subsequently used for clamping the ductus. The jaws have been ground so that they are thinner than stock instruments.

and the left main bronchus. After a hole has been made behind the ductus in this manner, the opening can be carefully enlarged by sharp dissection under direct visualization. Behind the back wall of the ductus there is usually a web of fibrous tissue which fans downward to the pulmonary artery, medially to the bronchus, and superiorly to the medial surface of the aorta. This substance

must be cut away adequately to give as much room as possible on the medial surface of the ductus. This dissection brings one dangerously close to the pulmonary artery, the left main bronchus, and the undersurface of the aortic arch, but in no case of the present series was any one of these structures injured.



Figs. 4, 5, 6.—¼, Ductus completely uncovered. Two clamps applied. 5, Four clamps in place. Ductus being cut between the two middle clamps. 6, Ductus completely severed, leaving two clamps on either end.

Touroff[6] has emphasized that sometimes it is possible to get into a tissue plane which allows the operator to dissect more freely around the ductus. All of the ductus dissection and liberation must proceed with great caution and must be completed with thoroughness, because this single step is most important in facilitating subsequent stages in complete division of the vessel.

Fig. 3 represents the type of instrument which has been used for clamping of the ductus. This is a standard Crile clamp, 5½ inches in length, the jaws of which have been ground off on both sides so that the surfaces are nearly parallel and are thinner than stock instruments. In the first eighteen cases in which complete division of the ductus was performed, each of these clamps was fitted with a rubber band on the handle so that the instrument could be placed on the ductus and held in that position by the tension imparted by the rubber band. This precaution was taken so that there would be no undue crushing injury to the ductal wall. In all of the subsequent cases, this elastic band has been discarded and each clamp has been placed on the ductus so that only its first ratchet is engaged. In no instance has this caused any important trauma to the ductus wall, nor has it led to subsequent sloughing or bleeding in any patient.

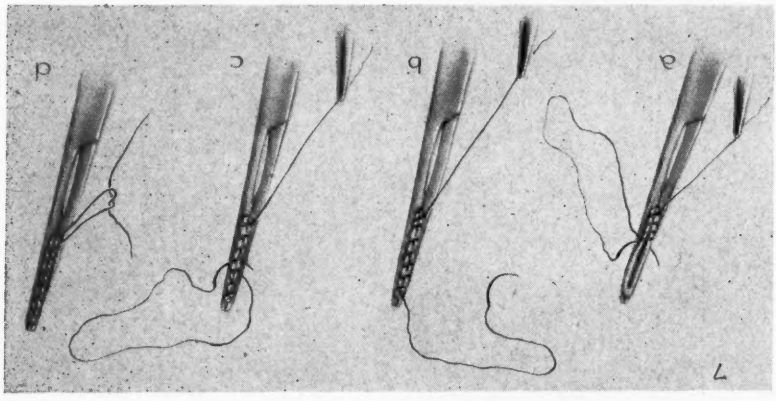
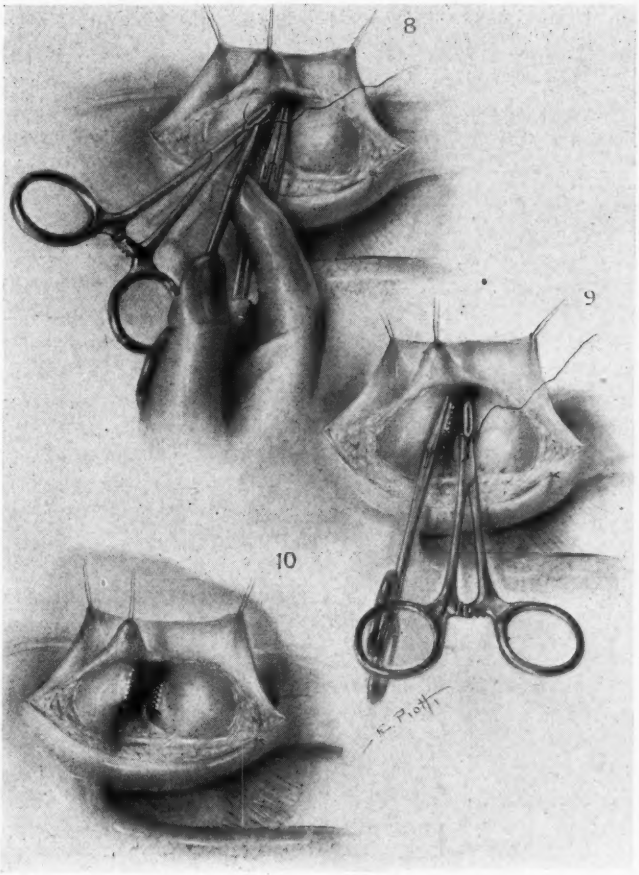


Fig. 7.—Details of closure of pulmonary end of ductus. One clamp has been removed, leaving a cuff which is sewed over and over with fine silk suture.

After the ductus has been completely freed of areolar tissue and has been cleared of any investing membrane, two clamps are applied as shown in Fig. 4. In most patients this appears to take up all of the room between the aorta and the pulmonary artery. However, it is possible (Fig. 5) to squeeze on a third clamp which rides up onto the pulmonary artery and a fourth clamp which rides up onto the aorta. With these four clamps in place, the ductus is cut in half by a scalpel which is run between the two middle clamps. This, then, leaves (Fig. 6) the divided ductus with two clamps on its pulmonary end and two clamps on its aortic end. It is at this stage that the above-described dissection will pay dividends, because the previous freeing of structures medial and posterior to the ductus will now allow the pulmonary artery and aorta to separate from one another and give the operator room to work on the individual ends of the severed ductus.

Fig. 7 shows details of closing the pulmonary end of the ductus. The presenting clamp (the one nearest the operator) has been removed and thus provides a small cuff of ductus wall which is 2 or 3 mm. in length. This can be

whipped over and over with a continuous 5-0 Deknatel silk suture carried on a small atraumatic needle. When the end of the ductus has been closed in this way, the remaining end of thread is brought back and tied to its original end.



Figs. 8, 9, 10.—*8*, Cuff on pulmonary end of ductus being closed in the manner shown in Fig. 7. *9*, Pulmonary end of ductus closed. One clamp is then removed from aortic end and the cuff of tissue will be sewed over and over, the stitch passing through the full thickness of ductus wall. *10*, Division of ductus completed. Individual closure of aortic and pulmonary ends of ductus.

Fig. 8 demonstrates the step of closing the pulmonary end of the ductus after the presenting clamp has been removed and a cuff has been made. In Fig. 9 a similar treatment is being carried out for the aortic end of the ductus, from which the uppermost clamp has been removed. The cuff thus provided is being sewn over and over with a continuous suture in a manner similar to that which was used for the pulmonary end of the ductus. After the suturing of pulmonary and aortic ends has been completed, it is customary to squeeze a gauze sponge

down between the aorta and pulmonary artery, and while this gauze is in place, the remaining pulmonary clamp is removed. The pack is held in place for several minutes to allow blood to coagulate between the interstices of the sutured end of the ductus. The pulmonary end is then inspected, and when it is found to be completely dry, another gauze pack is jammed in between the aorta and pulmonary artery and the remaining aortic clamp is removed. This pack is also held in place for several minutes to allow clotting to occur between the stitches in the aortic end of the ductus. The pack is then removed, and in the vast majority of cases the field is completely dry. In only a few cases has it been necessary to take an extra stitch or two to close some pinpoint opening. (In the first eighteen cases of this series, a second line of sutures was employed to bring together the adventitia of the pulmonary artery to re-enforce the first layer of sutures. A similar adventitial closure was likewise done on the aortic side. Later experience has shown that these second layers are not necessary.) Fig. 9 shows the condition of the aorta and pulmonary artery after all instruments have been removed and the two ends of the ductus have been individually closed. It is probably not essential to do so, but it has been my routine to close the mediastinum by repairing its parietal pleura with interrupted silk stitches.

It is unnecessary to enumerate here the steps for closure of the thoracic wall wound, which steps have been amply treated in a previous communication.[4] It is, however, well to emphasize that careful closure of the chest is of extreme importance in reducing the postoperative discomfort of the patient to a minimum, in producing a cutaneous scar which is not unsightly, and in reducing the postoperative wound complications to a negligible figure.

COMMENTS

In a group of ninety individuals who have been treated by complete division of a patent ductus arteriosus, the ages ranged from 2 to 46 years. In this series there have been two surgical deaths.* One fatality occurred two weeks after operation from a staphylococcus mediastinitis and overwhelming infection. The second occurred in a 26-year-old woman with an advanced stage of cardiac decompensation; division of the ductus had been completed, but the patient expired shortly thereafter with cardiac dilatation and failure. Thus, there have been no deaths which are attributable to ductus division itself. Hence, in the experience of this clinic, complete division of the ductus has not carried any higher operative risk than ligation of this vessel.

Without question, some individuals from this group of ninety patients could have had a perfectly satisfactory result from ligation. Possibly it would have been good judgment to ligate those ducti which were small, soft, and easily compressible, reserving the division technique for the more difficult cases wherein ligation would probably fail to give a complete and permanent closure. However, from a practical point of view, it has been better to use only one technique so that the surgical team could be accustomed to a single, routine procedure, and could standardize this thoroughly. In this way the operator and team have

*See addendum.

adequate rehearsal, and hence are better equipped to tackle the occasional case in which there is a formidable problem because of the conditions in the ductus or the regional vessels.

In these ninety individuals with complete division of the ductus, there were three with superimposed *Str. viridans* infection. While division was undertaken with some trepidation because of the friability of the walls of the pulmonary artery or aorta, in no case did hemorrhage occur at the time of operation or subsequent thereto. (In the total series of 133 patients with ligation or division of a patent ductus arteriosus, there were eleven instances of superimposed, active *Str. viridans* infection. These were all in the prepenicillin era. In no case could the blood stream be sterilized preoperatively with the sulfonamides which were then available. Of these eleven patients, four continued to have bacteremia following operation and subsequently died from continuing infection. The other seven have been cured of their disease.)

There are doubtless some surgeons who will maintain that division of a patent ductus carries an unwarranted risk, and that adequate results have been obtained by ligation alone. I would like to anticipate these objections by pointing out that personal communications from a considerable number of physicians and surgeons indicate that there are postoperative recurrences in a considerable number of patients. This information has not yet appeared in publications. Furthermore, any claims for completely satisfactory results following ligation should clearly indicate what types of cases have been operated upon. I fully agree that in children, where the ductus is soft, yielding, elastic, and easily compressible, a ligation can almost always be relied upon to produce a permanent closure. However, in older individuals, particularly in adults beyond 18 to 20 years of age, the ductus is frequently unyielding, is difficult to compress, and has an exaggerated pulsation transmitted to it by a sclerotic and fixed aorta. Under such circumstances I am convinced that ligation in any form will occasionally lead to partial recurrence of the fistula. It is primarily because of some disappointments in the adult group that I have turned to division of the ductus in all cases.

SUMMARY

The surgical technique herein described for complete division of the patent ductus arteriosus has been employed in ninety cases. Two patients died, one from staphylococcus mediastinitis and the other from cardiac decompensation. Neither fatality was related to the division itself, and in neither instance would simple ligation have avoided a fatal issue. The complete division of the patent ductus has been so completely satisfactory that all forms of ligation of the vessel have now been completely abandoned.

REFERENCES

1. Blalock, A.: Operative Closure of the Patent Ductus Arteriosus, Surg., Gynec. & Obst. **82**: 113, 1946.
2. Gross, R. E.: A Surgical Approach for Ligation of a Patent Ductus Arteriosus, New England J. Med. **220**: 510, 1939.
3. Gross, R. E.: Surgical Management of the Patent Ductus Arteriosus; With Summary of Four Surgically Treated Cases, Ann. Surg. **110**: 321, 1939.

4. Gross, R. E.: Complete Surgical Division of the Patent Ductus Arteriosus; a Report of Fourteen Successful Cases, Surg., Gynec. & Obst. **78**: 36, 1944.
5. Touroff, A. S. W., and Vesell, H.: Subacute Streptococcus Viridans Endarteritis Complicating Patent Ductus Arteriosus: Recovery Following Surgical Treatment, J. A. M. A. **115**: 270, 1940.
6. Touroff, A. S. W.: Further Experiences in the Surgical Treatment of Subacute Streptococcus Viridans Endarteritis Superimposed on Patent Ductus Arteriosus, J. THORACIC SURG. **12**: 1, 1942.

ADDENDUM

July, 1947. This series has now grown to 180 cases of complete division with a total of four deaths: one from *Staphylococcus mediastinitis*, one from faulty anesthesia, two from cardiac failure. There have been no deaths from hemorrhage, either at operation or subsequent thereto.

DISCUSSION ON "COMPLICATIONS OF THE SURGERY OF PATENT DUCTUS ARTERIOSUS" BY DR. JOHN C. JONES* AND "COMPLETE DIVISION FOR THE PATENT DUCTUS ARTERIOSUS" BY DR. ROBERT E. GROSS

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—When I was here last in 1939, I had an opportunity to see the first of these cases of Dr. Gross in Boston, and they really intrigued me. Upon returning, I talked to our physicians in Stockholm, and they were not enthusiastic over sending such patients for operation at that time, so it took me about two years to persuade them to try it.

At that time there was already one slight recurrence among Dr. Gross's patients. One could hear a continuous murmur, which was very much reduced compared with the murmur that the child had before operation. We discussed the possibility of dividing the ductus because of this recurrence at that time. In my first operation, a rather wide and short ductus, I divided. You have seen the technique, which was very similar to the one Dr. Gross has described to you just now.

The next few patients had rather long and narrow ducti which I did not consider it necessary to divide. I ligated them doubly and had about 4 or 5 mm. of the ductus between the two ligatures, one up on the wall of the pulmonary artery and the other close to the aorta. I hoped that scar tissue would develop.

Since that time we have operated upon seventy-one patients, and have found that a little more than half of them have had ducti which had such a tough wall and were of such length that they could be ligated easily and safely. The other half, on the other hand, had a short and wide ductus, and those we have divided.

In a borderline patient with not very good general condition, it seemed possible to ligate the ductus, and, wishing to get out of that chest as soon as possible, I did a ligation. When the ligature was tied, it cut through, and bleeding from both the pulmonary artery and the aorta occurred at the same time. It was somewhat difficult to control the bleeding, but it was possible. It was necessary to apply clamps across the aorta above and below the hole in the aorta and to apply a side-clamp to the pulmonary artery.

When that was done, the bleeding was controlled, and it was possible to suture the aorta and the pulmonary artery.

In all the other patients I have used that technique which was forced upon me the first time in this case.

Now we have somewhere between thirty and thirty-five successful cases in which we have used the cross-clamping on the pulmonary artery.

It is possible to excise all the ductus tissue out of the aortic wall, and get normal aortic wall edges sutured against each other, which may be more safe than to leave tissue behind which might later give difficulty in forming aneurysms. In our whole series,† we have had only one recurrence, and that was in a divided septic patient.

*See page 305 for article by Jones.
†Seven with sepsis; all the other ones without infection.

This was an infected patient in which I divided the ductus. She seemed to do quite well at first, blood cultures were negative, and she was sent home as cured. Two or three months later she came back to the hospital with the same condition she had had when she first entered. I tried to reoperate, and at operation it was found that a sort of aneurysm had developed between the aorta and the pulmonary artery, and there was a wide communication between the two vessels.

Since then I have used the pericardial flap, which always covers the ductus as a sort of plastic material to suture down in the bottom of the groove between the pulmonary artery and the aorta after division of the ductus.

I quite agree with Dr. Jones that it is advisable to have a very good approach in cases like this. I have used the same incision shown you here today, for the ductus, usually taking away the fifth rib, sometimes the fourth.

If bleeding complications should occur during operation it is essential to have a very wide incision to be able to control a severe bleeding.

We have had in our series, two deaths. The first one was the patient with the recurrent condition, just described. She died on the operative day after we had tried to close her fistula once more. We could not close it at operation, and she expired a few hours after operation.

Another patient who died was a little child, about 5 years of age. She had one of the shortest operative times that we have had, and everything was quite uneventful. When the operation was finished, an aspirating tube was passed through the intratracheal tube, and she was aspirated with too high a negative pressure. I didn't observe that the catheter passed through the intratracheal tube was passed with difficulty, so I believe that the sudden death must have been due to a very rapidly increased negative pressure inside the bronchial tree. She had an acute heart stop. We re-entered and massaged the heart, and were able to get her back to life, but the time it took was too long. She developed cerebral symptoms, and died within twenty-four hours after operation.

I would be a little afraid of tearing and pulling with the type of clamps Dr. Gross showed just now. On these heavy clamps, you do not need to have much movement in order to tear a fragile ductus and have bleeding. It must be necessary to be very careful in using those clamps, and it might be safer to use cross clamping of the aorta and side clamping of the pulmonary artery.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—My associate, Dr. Varco, and I want to record a minor variation in the technique of closure of the patent ductus arteriosus.

All of us are aware of the great impetus Doctor Gross has given thoracic surgery in developing his own interest in vascular sugery. In Minneapolis, there has been an intense interest in the subject of patent ductus for a long time, but we, as our Chairman put it yesterday, had our eye on the sight but we failed to pull the trigger.

In 1935, Dr. George Fahr, well-known Minneapolis internist, had a patient of 22 years with a patent ductus for whom he recommended the operation since done by Dr. Gross. Dr. Elliott Cutler in February of that year came out to Minneapolis to give the Judd Lecture. Dr. Cutler, Dr. Fahr, and I discussed the problem at length and subsequent to Dr. Cutler's visit, he and I exchanged letters upon the matter. Dr. Cutler's parting advice to me upon the matter, as I remember it, was to put a Parham band upon the ductus.

Our patient had a large heart and cardiac decompensation. As I recall it, Dr. Gross's first patient was 7 years of age. The experience that my associates and I have had in operating upon patients past late adolescence suggests that the operation in such individuals is ordinarily more difficult than in the younger age groups. It was providential that Dr. Gross chose for his first trial with the procedure a young child, in whom the problem of dissection was simpler.

Since Dr. Gross's description of division of the ductus, Dr. Varco and I have followed this procedure exclusively. We have had the experience of recanalization following ligature of the ductus, which is not pleasant.

(Slide.) We have now divided the ductus in thirty-three consecutive cases without deaths or recurrence; in fact, we have given up ligature altogether. The special point which we wish to emphasize in the dissection is the development of a tissue plane to facilitate the dissection at its most difficult point, the anteromedial angle between the aorta and the pulmonary artery. The fibrous layer of the pericardium is tougher than the walls of the two adjacent vessels, which the dissection proposes to separate. In consequence, this part of the dissection is always slow and difficult. In some instances, as Dr. Gross has been at pains to point out, the lappet of pericardium over the ductus may be dissected off readily. Many times, however, such is not the case, In those instances, we define the outer, tougher, fibrous pericardium and dissect it off the thin, inner, serous pericardium. Failing to do this readily, we open the pericardial sac deliberately and free the ductus from the outer fibrous layer of the pericardium. In the presence of a large heart and a large pulmonary artery, especially in older children or adults, this dissection is not easy. Nevertheless, we feel, after having employed this scheme routinely in all patients in whom the ductus has been divided, that this expedient simplifies the most difficult part of the dissection. Having dissected off the fibrous pericardium, the separation of the aorta from the pulmonary artery on their medial aspects becomes now an easier dissection. In the final stages of the dissection, employment of an illuminated lucite elevator or dissector has proved very helpful in identifying the diaphanous fascial layer before the aorta and pulmonary artery, after the fibrous pericardium has been dealt with.

In the division of the ductus, we employ three long, straight hemostats. Two are put ordinarily on the pulmonary artery side and one on the aortic side of the ductus. The wider cuff is left on the side with the single clamp. The upper clamp on the pulmonary side is removed and the cuff oversewn, as described by Gross. Before removing the remaining hemostat, however, we place a "stick-tie" suture behind the clamp, the suture being anchored in a remnant of fibrous pericardium. This scheme insures satisfactory hemostasis without bleeding. It is important not to place this suture in the arterial wall, lest the lumen of the vessel be entered, thereby inviting troublesome oozing. The isolation of a remnant of fibrous pericardium in juxtaposition to the remaining hemostat, on the pulmonary artery as well as aortic side of the ductus, affords an excellent anchorage for the "stick-tie" suture which will insure satisfactory hemostasis.

We have employed this method in the instance of a boy of 20 with a large, broad communication between the pulmonary artery and the aorta in which the two vessels appeared to fuse without the intermediary of a connecting ductus.

DR. ARTHUR S. W. TOUROFF, New York, N. Y.—Since returning from the Army, I have operated upon five patients within a relatively short period of time; and accordingly, assume that there are still many patients suffering from patent ductus arteriosus, who will sooner or later come to surgery. The subject of ductal surgery, therefore, remains timely.

My experience with ductal surgery began early in 1940, when I had occasion to operate upon four patients suffering from patent ductus with superimposed subacute bacterial endarteritis. Some of you may recall that the results were reported at the 1940 Meeting of this Association and that they were not particularly brilliant. Among the four patients, there was one cure of the infection, two deaths from hemorrhage, and one in which operation failed to influence the course of infection.

Subsequently, with increasing knowledge of the problems presented by operating in the presence of infection, the results improved. By the time I entered the Army in 1942, I had operated upon nine additional patients with infected patent ductus. Of these, seven were cured and two unimproved. There were no operative deaths. Today, with the use of massive doses of penicillin, it can be assumed that there will be little necessity for operating in the presence of active infection. However, our results did establish conclusively that surgery alone was curative. I might add that our eight cured patients have been followed for from four to six and one-half years and that all of them have remained well. This, in turn, would indicate that operation tends to protect against the development of infection.

Both Dr. Jones and Dr. Gross have mentioned cases in which they assumed that recanalization of the ductus occurred after ligation. While in some of their cases the patient developed unmistakable evidence of recanalization as evidenced by typical changes in blood-pressure, in others the only evidence on which the diagnosis was made was the persistence of a murmur. I do not believe that the mere presence of a murmur, without the other criteria of ductal patency, indicates that the ductus is open.

In the first patient with patent ductus complicated by superimposed endarteritis there was serious operative hemorrhage. Fortunately, the hemorrhage was controlled but, in the process, the ductus was doubly clamped, completely divided, and ligated. We had a sterile stethoscope available during operation and listened to the murmur periodically. It was interesting to note that even after the ductus had been divided and ligated, a loud murmur persisted over the pulmonary artery. When the pulmonary artery was compressed, the murmur disappeared only to reappear when the pressure was released. I assumed that the murmur was produced by the swirl of blood through the dilated pulmonary artery. An analogous situation obtains in cases of congenital dilatation of the pulmonary artery. In such cases, a characteristic murmur often is present. In the case which I have cited, the murmur persisted for several months despite the fact that the circulatory status became normal promptly. I have encountered similar situations on a number of occasions since then and, therefore, am not inclined to accept a diagnosis of postoperative ductal patency merely on the presence of a persistent murmur.

In those instances in which a ductus actually is patent following operation, patency may be attributed to incomplete occlusion by the ligature at the time of operation or to sloughing secondary to infection following operation. Both Dr. Jones and Dr. Gross have stated that in a number of their cases they tied their ligatures rather lightly for fear of cutting through the ductus. Furthermore, I believe that Dr. Gross originally recommended the use of cellophane as a ligature material because he feared a tight silk ligature and deliberately sought some material which would produce a substantial degree of periductal fibrosis and thereby result in gradual complete occlusion of the ductus. I, personally, have tied two or three heavy silk ligatures about the ductus as tightly as possible. In none of the cases has there been evidence of cutting through of the ligatures following operation, despite the fact that a great deal of force was applied in tying them.

Assuming that a ductal ligature has not been tied loosely, it is my impression that recanalization is the result of cutting through, induced not by tying the ductus too tightly, but by infection. Furthermore, I believe that secondary infection also is responsible for late hemorrhage such as has been reported by Dr. Jones. Although Dr. Jones's patients demonstrated no clinical evidence of infection, it seems fair to assume that the granulation tissue and the pulmonary erosion which were found at the time of reoperation could only have been the result of continued low-grade infection. In view of the time interval which had elapsed between the first and second operations, one would expect to find firm scar tissue everywhere in the local operative area. Instead, Dr. Jones found soft granulation tissue and a local erosion of the lung, although cultures proved sterile. I would like to ask Dr. Jones whether the patient received penicillin or sulfonamides, and if so whether they might not have been responsible for the sterile cultures?

In regard to ductal division, I have had experience in only three cases. These were all early in my experience with ductal surgery. None of them was planned and all of them were done in an effort to control accidental hemorrhage. Although first to perform ductal division, you now can appreciate why I have long considered it a doubtful honor. While overseas, I received from Dr. Gross a reprint of the article in which he first described his technique of planned ductal division. In view of the operative findings previously encountered in certain cases, notably adults, I wrote him that I would be very much interested in hearing of his future experiences with the operation. In operating upon adults, I often found the ductus to be sclerotic, thinned out, and sometimes calcific. The pulmonary artery usually was much more dilated and thinner than in children, apparently because of the fact that the lesion had been of longer duration. Not infrequently, the sclerotic ductus was firmly adherent to the

thinned-out, dilated, pulmonary artery. I found that such a state of affairs was much more likely to be attended by accidental operative hemorrhage than in cases in which the ductus was soft and rubbery and had much more nearly the consistency of a normal vessel.

I would like to ask Dr. Gross whether he has been able to divide the ductus as safely in adults as in children and whether he has specifically applied the method in cases in which the ductus was obviously sclerotic and rigid. On theoretical grounds, it would appear to be more hazardous because of the poorer tendency of the rigid-walled ductal ends to be apposed by sutures and of healing to take place. However, Dr. Gross's experience may indicate that the danger of ductal division in adults is more theoretical than real.

In closing, I would like to say a few words in regard to ductal division in infected patients. Dr. Crafoord referred to a case in which he attempted to divide an infected ductus, and regretted it. His experience would tend to support my contention that, in the presence of infection, healing is poorer and complications apt to occur. In the presence of infection, it therefore appears wise to avoid ductal division if possible. If division should become necessary as a result of hemorrhage, I would recommend the avoidance of any operative technique which requires the introduction of a considerable amount of suture material, and the use of two, simple, fairly heavy silk ligatures which are placed as far back on the ductal ends as possible. In the three cases in which I had occasion to perform ductal division in the presence of infection, this method was used and was followed by uneventful recovery.

DR. EDGAR W. DAVIS, Washington, D. C.—I wish to call your attention specifically to one patient, a 17-year-old boy with a moderately enlarged heart upon whom surgery was performed. The duct was found to be about 8 mm. long and only about 6 mm. in diameter. Two ligatures of cotton tape were easily applied around the duct and were tied tightly. About 50 per cent of the murmur persisted.

Dr. Touroff's original report described the persistence of a murmur in one patient in which the duct had been divided. Dr. Crafoord has just stated that in one of his patients, in whom the duct was divided, the murmur persisted. In Dr. Blalock's series, the only persistent murmur was in one in which the duct had been divided. These reports, therefore, make one wonder if there is possibly some other source of the murmur. With this in mind, I advised another operation for the patient mentioned above.

Three weeks following the initial surgery, the second operation was performed. The ductus arteriosus was surprisingly short and of such small diameter that it was almost inconceivable that it could be patent. In fact, the duct was found with difficulty and then only after finding the knot of ligature which had been applied at the first operation. The duct was then isolated and two more ligatures of umbilical tape were applied, one on top of the other, because the duct was too short to apply them side by side. Ten days later, when the patient was discharged, a murmur was not heard. Six weeks later, when he returned for a checkup, a murmur was present. Although the patient has a persistent murmur, he is clinically cured, as evidenced by the normal size of the heart and his ability to engage in competitive sports. I doubt seriously that this duct, with four ligatures around it, all of them tied tightly, could be patent.

Our series consists of twenty-six operations performed upon twenty-five patients. Our youngest patient was 22 months old and the oldest was 44 years of age. Both infected and noninfected patients are included. Complications consist of temporary hoarseness in one instance which cleared in a week, and the aspiration of bloody fluid from the first three patients. Since then, no thoracenteses have been done. The mediastinal pleura has been closed in the last three patients. No deaths attributable to surgery have occurred in this series. One death occurred ten weeks after operation, from intravenous heparin and penicillin given continuously. The family physician suspected subacute bacterial endarteritis, but could not prove the diagnosis. No murmur was present just prior to death. An autopsy was refused. With the exception of the patient who was operated upon twice, none had either a persistent or a recurrent murmur.

After we had used double ligation in fourteen cases of patent ductus arteriosus, we decided to divide and suture in the next fourteen patients and then compare the results of the

two methods. However, when we studied our first fourteen cases and found that we had such good results, we did not feel justified in changing from a very simple procedure to one which is obviously hazardous and technically much more difficult.

I am not attempting to be a "balance wheel," neither am I opposed to the "division and suture" method as advocated and so ably used by Dr. Gross. If, in subsequent years, we find that the division and suture method can be done with the same degree of safety, with the same or lower mortality rate, I am all for it. However, until then, those of us who perhaps are not the masters of vascular surgery, as Dr. Blalock, Dr. Crafoord, Dr. Gross, and a few others, will find it a much safer procedure to do a double ligation with No. 7 cotton tape, than to divide and suture.

DR. JOHN JONES, Los Angeles, Calif.—Our severest critics, the cardiologists, use two criteria for establishing a diagnosis of the recurrence of the murmur. One is that when they hear a continuous murmur over the pulmonic area, they assume that the fistula has either re-established or that the ductus was never closed off completely at the time of ligation. The other is the sound track. Those two we have depended upon and done routinely, and, if we detect a continuous murmur in the sound track, we assume that the fistula has re-established or the fistula was never closed off.

The remark about having proved that dilated pulmonary artery makes me feel better. Perhaps some of mine that recurred were that, but we must go on the assumption when we hear the murmur when we take the dressings off a day, or two, or three, or a week after operation, that probably the ligation wasn't tight enough.

In the case that you mention, Dr. Touroff, about there having been a possibility of infection at the site of ligation that caused the fistula between the aorta and the lung, the material was examined by the pathologist, and he reported fibrous tissue, blood clot in various cases of organization formed by reaction, and no evidence of inflammatory reaction in this tissue removed; so I must assume that it was not an infection and was formed by the reaction and erosion by the tapes.

If we had had an inflammatory reaction or an infection at the site of the ligation, we would have had the same experience as in an early case reported in Los Angeles, where we had an endarteritis, and vegetation was both on the pulmonary side and the aortic side of the vessels in autopsy.

DR. ROBERT E. GROSS, Boston, Mass.—Dr. Touroff asked about the ages of patients and the question of performing divisions in adults. I should have mentioned that our patients varied from 2 years to 46. I would have been hesitant at first about undertaking divisions in adults, but I think that if you gain experience in youngsters, where it is easy, you can carry out the procedure in adults.

Regarding the question of division in infected patients, we have operated upon eleven infected patients. Three came within the period in which we have been doing divisions, and with some trepidation division was undertaken. In no instance was there any bleeding or any untoward difficulty. I did, however, take the precaution of placing something between the aorta and the pulmonary artery. This was a flat piece of pectoral muscle fascia, about 2 cm. square, which was interposed between the aortic and pulmonary artery suture lines. Dr. Wangensteen indicated that pericardium can be pulled between the suture lines. This seems to me to be an admirable suggestion.

LUNG RESECTION FOR PULMONARY TUBERCULOSIS

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PULMONARY resection is a relatively new type of treatment for tuberculosis, at least in its modern form. As yet it is under some cloud because of the dramatically poor results first obtained. However, most new procedures, methods, and mechanisms have certain inherent bad features which can be eliminated only by persistence and increasing experience. After an experience with eighty pulmonary resections for tuberculosis, I feel satisfied that the procedure is here to stay, will become progressively standardized, and will become increasingly more satisfactory as regards long-range "cures."

First, it is painfully apparent to every phthisiologist that collapse therapy is unavailing or actually dangerous in certain types of tuberculous involvement such as tuberculoma, stenosis of large bronchi with definite retention of secretions, and persistent check-valve or tension cavities, and should never be utilized in them. To this list might be added cases of unilateral, caseous pneumonia with extensive cavitation throughout an entire lung (the "destroyed lung"). In addition, there are many cases in which collapse therapy has already failed repeatedly, or in which it will probably fail. These are cases in which satisfactory anatomical collapse by thoracoplasty has failed to convert the sputum (the same with pneumothorax) and certain cases of lower lobe cavitation, giant cavities, and residual tuberculous bronchiectasis.

It is evident that there is a very definite need for some effective type of treatment in such cases. Pulmonary resection would seem to be the answer, providing it can be done with an acceptable mortality and morbidity. It must be noted that this mortality and morbidity does not have to be comparable to that in thoracoplasty or pneumothorax therapy, since resection is recommended only in cases unsuitable for these treatments. Of course, if and when the mortality and morbidity of resection is reduced to levels comparable to that of collapse therapy, then some consideration may be given to generally replacing collapse measures by extirpation of the patient's most malignant tuberculous focus.

It is essential to remember that tuberculosis is always a generalized disease, with demonstrable foci in lungs, kidneys, spleen, bones. One must not forget that we can only hope to extirpate or heal the most malignant tuberculous lesion, whether it be by lobectomy, thoracoplasty, nephrectomy, arthrodesis, spine fusion, or amputation of an extremity. We trust that the patient will then be able to heal all other foci in his body. Hence, he still must be under

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a general therapeutic regime for a prolonged period of time. It must be fully recognized that if the patient is suffering from a progressive generalized or even multiple focus tuberculosis, our local efforts are doomed to eventual failure except in rare instances. Thus pneumonectomy for acute caseous pneumonic disease bears a much poorer eventual prognosis than pneumonectomy for severe bronchial stenosis with essentially healed parenchymal disease, since in the former case the generalized disease process is much more likely to produce another virulent focus in the other lung or elsewhere. This consideration does not necessarily contraindicate pneumonectomy in acute pneumonic disease, but it does indicate that the eventual prognosis and statistical results in such cases must inevitably be poor.

Having mentioned the generally recognized indications for pulmonary resection in tuberculosis, and having suggested the possible results obtainable by technically successful operations, we must consider in some detail the methods by which technical success may be routinely obtained.

The commonest causes of mortality and morbidity in resection for tuberculosis are: (1) asphyxia during the operation due to mechanical expression of copious secretions into the bronchial tree of the nonaffected lung; (2) spread of the disease to the contralateral lung or to the remaining portion (if any) of the same lung; (3) hemorrhage or shock; (4) postoperative opening of the bronchial stump, producing a bronchopleural fistula; (5) tuberculous empyema, early or late; (6) pyogenic empyema; (7) a combination of the last three complications; and (8) marked departures in postoperative pleural pressures from the normal.

Let us take up each consideration in detail.

1 and 2. Prevention of obstruction of the airways by copious secretions from the affected lung is extremely important. In the usual lateral or posterolateral operative approach, and even the anterolateral approach, gravity insures that any secretions expressed from the diseased lung must pass into the trachea and then enter the bronchial tree of the more dependent lung. The anesthetist may be able to aspirate part of these secretions with a catheter introduced through an endotracheal tube. In cases where the secretions are copious, he must inevitably fail to remove all of them, since the tracheal aspirations must be interrupted by longer periods during which anesthetic gases are introduced. Once these secretions enter the smaller bronchial branches, they can only be removed by coughing, since even the bronchoscopic aspirator passed under direct vision cannot evacuate the contents of obstructed bronchioles. Add to these mechanical factors the reluctance of many anesthetists to aspirate through the endotracheal tube unless audible bubbling of secretions is present, and one will see that such nonchalance in surgical approach and anesthesia must be attended by considerable risk of actual asphyxia, and, where this is avoided, of obstruction of the smaller bronchi by infectious secretions. This latter situation leads in many cases to segmental atelectasis and tuberculous pneumonitis ("spread").

One would think that endobronchial anesthesia would prevent the development of asphyxia or contralateral tuberculous spread. Unfortunately, the

usual type of endobronchial anesthesia tube is arranged to be passed blindly. Thus, one cannot be completely sure that the tip of the tube is in the appropriate bronchus, or how far in. In an attempt to improve this method, Dr. Henry S. Ruth and myself have modified a Woodbridge-Flagg type of tube so that the obturator is really a 5 mm. bronchoscope. This may be simply and accurately

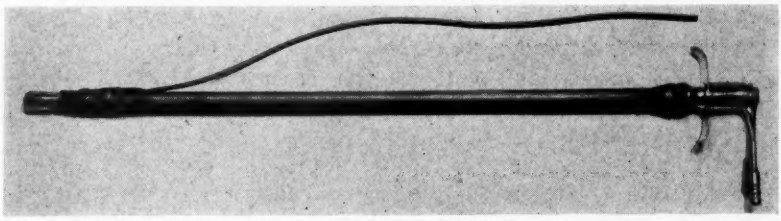


Fig. 1.—Direct vision endobronchial catheter, obturator in place. The rubber sheath has been drawn back from the tip to show the coil-spring construction. Note inflatable cuff. Obtainable from Richard Foregger Co., New York City.

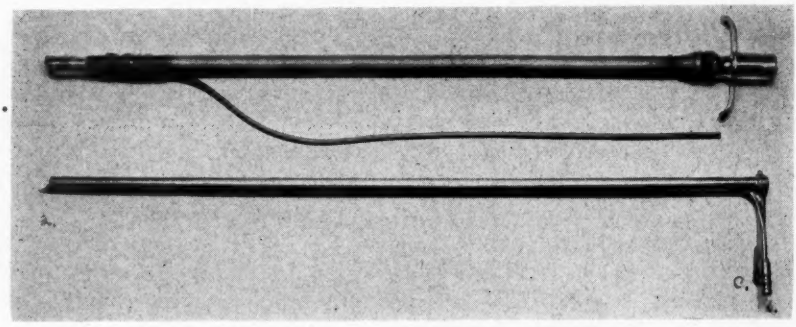


Fig. 2.—Obturator removed. It is really a 5-mm. bronchoscope with a built-in light carrier (see bulb-a) and oxygen delivery tube (b). Battery cord is connected to terminal (c).

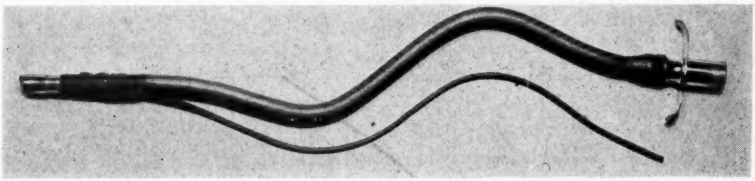


Fig. 3.—Flexibility of tube after removing obturator. This lessens trauma to air passages.

passed into the bronchus of the normal lung for the desired depth. An inflatable rubber cuff close to the tip obstructs passage of air or mucus between the trachea and the intubated bronchial tree. (See Figs. 1, 2, and 3.) A very satisfactory endobronchial anesthesia is thus obtainable and contralateral spread or asphyxia from secretions would seem to be preventable by this method. However, sometimes the inflatable cuff may become deflated. Therefore, it is advisable to employ a considerable degree of Trendelenburg position, so that

gravity will aid in keeping secretions from passing by the possibly deflated cuff. Another factor in this type of anesthesia is the frequency with which the right upper lobe bronchus is obstructed by the inflated cuff. This permits patients undergoing left-sided surgery only the right middle and lower lobes for oxygenation of their blood. In the hands of a good anesthetist, no difficulty will be observed during such anesthesia, but considerable aid by manual compression of the anesthesia bag may be necessary.

In cases of lobectomy or partial lobectomy, endobronchial anesthesia offers no protection against homolateral spread of secretions, and it prevents periodic inflation of the lung being operated on, which may be important, especially in segmental surgery.

For these reasons, even having a satisfactory endobronchial tube, we use it only in cases of total pneumonectomy and lower lobe lobectomy. In upper lobe resection, middle lobe resection, and those total pneumonectomies which we expect to be technically easy, a modification of the Rienhoff anterolateral approach is used with the patient in an extreme Trendelenburg position on the operating table. The approach is really an anteromedial one through the third anterior interspace with or without resection of an adjacent rib. The good lung is elevated above the diseased one by a pillow under the shoulder of the unaffected side, or by tilting the operating table laterally. For such cases, catheter suction through a simple endotracheal tube provides efficient evacuation of secretions which either stay in the affected lung or lobe, or, if they do enter the trachea, do not cross to enter the upper lung.

The Overholt support, which permits a posterolateral type of operative approach with the patient in a slight Trendelenburg and the face down position, the disease (operative) side being tilted lower than the good side, appears to overcome most of these problems. In this position, a very large surgical exposure is obtained, while secretions are expressed out of the diseased lung only with difficulty, and then drain into the trachea and out the mouth by gravity. Gravity is assisted by suction through a simple endotracheal tube. Unfortunately, this method, too, does not completely prevent homolateral expression of secretions (homolateral atelectasis and spread).

3. Hemorrhage and shock in resection for tuberculosis does not differ from that seen in resection for other pulmonary diseases. It may be combated in the usual way by gentleness, meticulous hemostasis, infusions, and transfusions. The average pneumonectomy or lobectomy patient probably loses 1,500 c.c. of blood during the operation and the next twenty-four hours. This should be replaced while the patient is on the operating table or soon after. Some patients, of course, may lose much more blood.

4. Opening of the bronchial stump is now, fortunately, becoming rather rare. Almost every thoracic surgeon has his own method of bronchial closure and every one is getting improved results. Essentially, most thoracic surgeons have stopped expecting the bronchus to heal *per primam*. They merely close it with fine sutures which are placed so as to interfere as little as possible with

the intrinsic bronchial blood supply and yet securely prevent bronchial leakage. They then cover the cut end with some viable tissue to produce the effect of a blowout patch. Within a few days this tissue is presumably tightly healed so that when the sutures finally cut through the bronchial wall (in ten to fourteen days) the healed overlying tissue prevents a clinical bronchial opening. I have used many methods of bronchial closure. At the present time I am utilizing a row of end sutures of fine silk or stainless steel wire, passed through cartilaginous and membranous walls, and another row of mattress sutures about ¼ inch proximal to this. These mattress sutures pass around a ring of cartilage and through the membranous wall. Thus both rows of sutures are passed in line with the long axis of the bronchus, and consequently interfere very little with its blood supply. (See Fig. 4.) The bronchial stump is then sprayed with plasma and thrombin to produce a sticky layer of thin, clotted plasma. Any available adjacent tissues such as edges of mediastinal pleura are sutured accurately to the bronchus. This may usually be reinforced by an additional

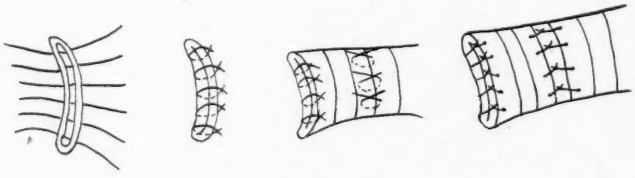


Fig. 4.—Method of suturing bronchus. Fine silk or stainless steel wire is used. Bronchial stump must then be covered by a viable tissue.

pedicled flap of parietal pleura. In instances where surrounding available tissues are scanty, I have recently been using Overholt's method of suturing a free graft of fascia from the subscapular region directly to and over the bronchus. In spite of early misgivings, there have been no bronchial openings or untoward incidents in eight cases so treated, even though minor pleural contamination was present in three instances. Each case has progressed uneventfully. Early in the history of pulmonary lobectomy it was customary for some surgeons to suture the bronchial stump to the residual lobe. This has been largely abandoned because it may hamper proper re-expansion of the remaining lobe, and also because attempting re-expansion of the lobe may tear out the sutures leading to air leakage or hemorrhage. However, if the remaining lobe can completely fill the hemithorax by re-expansion, it will automatically seal off the entire pleural space and adhere over the bronchial end or mediastinum, thus preventing any clinical bronchial opening. Therefore, suction drainage of the pleura is routinely employed after lobectomy and segmental pneumectomy.

5. Tuberculous empyema (simple) does not seem to follow soon after a clean pulmonary resection. Therefore, in a clean lobectomy or segmental resection with early re-expansion of the remaining lung tissue the pleural space can usually be completely obliterated. The lung becomes generally adherent to the parietal pleura and no empyema follows. Even if the pleura is contaminated

by operative rupture of a tuberculous cavity, provided the amount of contamination is reduced by suction and washing the pleura freely with saline, early, complete re-expansion of the remaining lung tissue seems to protect effectively against the development of a tuberculous empyema. For this purpose we use a catheter and a constant pressure drainage system (–15 cm. water) in all cases of partial pulmonary resection. Within twenty-four to forty-eight hours the re-expanding lung tissue usually has obliterated the entire pleural space. There is one exception to this favorable response. In cases where a cavity is ruptured and a part of the tuberculous wall is left attached to the chest wall, a large percentage of patients will develop a tuberculous empyema. This occurred in three of four such cases in the present series. Here, it might be emphasized that in pulmonary resection subsequent to thoracoplasty the risk of operative cavitary rupture is very high. Thus in twenty-five such cases there were fifteen cavitary ruptures (60 per cent). In fifty-five nonthoracoplasty cases only four cavitary ruptures occurred (5.4 per cent). While several of these patients were reported to have bacilli in their early postoperative drainage, only four developed a tuberculous empyema, the rest absorbing their fluid in a few days. Such cases often run a high fever for two to three weeks, which then subsides by lysis. The cause may be related to pleural tuberculinization.

In patients with total pneumonectomy such operative cavitary rupture is rare except in post-thoracoplasty cases, and we have had no experience with it. In post-thoracoplasty cases, performance of a phrenic paralysis at the time of operation and suction drainage usually permit early obliteration of the pleural space so that no empyema results. However, patients with clean pneumonectomy frequently develop a late (three to eighteen months) tuberculous empyema unless the pleural space is obliterated by thoracoplasty and permanent phrenic paralysis. Indeed, of eight clean pneumonectomies performed early in our series in which no subsequent thoracoplasty was performed, three patients developed late (three to eighteen months later) severe clinical tuberculous empyema with respectively: a chest wall abscess; a bronchial fistula (nearly a year after operation); and a bronchopleural-esophageal fistula (proved at autopsy). Two of the rest have developed late draining sinuses from the chest wall and presumably have at least small underlying tuberculous empyemas. It has been impossible to prove the latter because of lack of cooperation of the patient in one case, and the family physician in the other. The other three patients seem to be well enough, but this experience has led us since then to perform an obliterative thoracoplasty and a permanent phrenic paralysis routinely after every case of total pneumonectomy for tuberculosis. A partial thoracoplasty is performed after upper lobectomies, especially where the lower lobe fails to re-expand and fill the apex of the thorax.

6. Purely pyogenic empyema in these cases has practically disappeared since the routine employment of preoperative and postoperative penicillin therapy. It occurred in four of our early cases, and was probably related to an unnoticed breach of surgical technique. It tends to become mixed by the addition of tuberculous infection.

7. Opening of a bronchus into the pleural cavity ordinarily leads to a mixed tuberculous empyema, although either pure tuberculous or pure pyogenic infection may sometimes ensue. Either of them appearing primarily may predispose to opening the bronchial stump. This serious combination of complications is frequently fatal and requires rigorous management. While the routine employment of a thoracotomy, thoracoplasty, and finally, Schede type of program, may eventually salvage the patient's life, it is a prolonged and uncertain course. Perhaps we should boldly invade the tuberculous pleural space, and reamputate and reclose the bronchus, even if this should mean further pulmonary resection. Fortunately, such complications become rarer as time goes on, and may eventually become practically nonexistent.

8. Morbidity or death from pleural pressure changes may be related either to air leaks from the sutured bronchus or lung tissue, to pleural effusion or bleeding, or to homolateral shifting of the mediastinum due to excessive escape of pleural air or fluid from the operative pleural rent, which can seldom be closed in a truly airtight manner. (See Fig. 5.) In order to control these possible departures from the normal, most thoracic surgeons resort to frequent thoracentesis and to determination of the plueral pressure levels in the tightly closed chest. Some surgeons routinely drain lobectomy cases but depend upon thoracentesis to control pleural pressures in the total pneumonectomies. Various experiences, some very dramatic, have led the author to adopt tne policy of drainage in every case at a constantly maintained negative pressure (–15 cm.). In pneumonectomy patients, the drainage tube is removed in forty-eight hours. In lobectomy patients, a suction motor is used to exaggerate the negative pressure somewhat, and the tube is removed in three to four days, when postoperative x-rays have revealed complete lung re-expansion. (See Fig. 6.)

Besides the adequate management of each of these factors, there are certain other very important considerations which must be satisfied if routinely good results are to be obtained. First, the surgical technique must be meticulous. Individual ligation of the hilar vessels and careful closure of the bronchus are so essential that it may be said that a lung tourniquet should be on the operating table only to protect against an extreme emergency. It has no other use in pulmonary resection for tuberculosis.

Postoperatively, these patients should ordinarily be bronchoscoped before leaving the surgical theatre. Subsequently, great pains should be taken to maintain complete evacuation of the air passages. The cough mechanism is greatly impaired by the presence of a paralyzed diaphragm even after a total pneumonectomy. This is due to unstabilization of the mediastinum because of the atonicity and paradoxical movement of the operated diaphragm. Thus, in the inspiratory phase of the cough cycle (inspiration, compression, and forceful expulsion of air) the paralyzed diaphragm is drawn up into the thorax, permitting the mediastinum to be drawn toward the unoperated side. Thus, a full inspiratory filling of the remaining lung is not attained.

Similarly, diminishing the rigidity of the chest wall by extensive or multiple rib resection is attended by impairment of expectoration because of interference

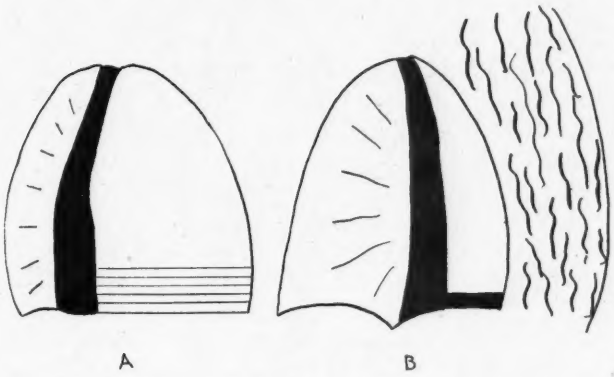


Fig. 5.—*A*, After pneumonectomy the heart and mediastinum may be forced toward the opposite side due to leakage of air or accumulation of fluid under pressure in the pleural space. *B*, After pneumonectomy air and fluid may escape during coughing from the pleural cavity through a pleural rent into the layers of the chest wall producing emphysema or ecchymosis. The very high negative pressure produced in the pleural cavity draws the mediastinum toward it causing torsion of the great vessels and overdistension of the remaining lung. Simple water-seal drainage may produce a similar high negative intrapleural pressure.

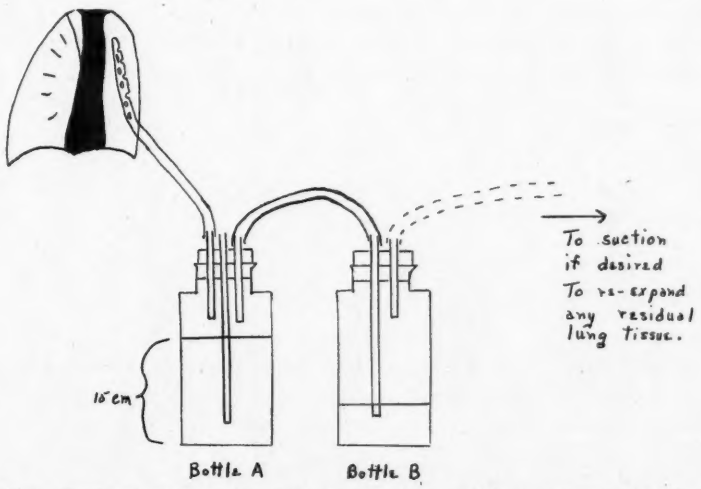


Fig. 6.—Constant pressure pleural drainage system. Bottle A prevents pleural pressure from becoming more negative than −15 cm. water (or whatever the height of the fluid column is). Bottle B is a simple water-seal trap to prevent positive pleural pressure from developing. To re-expand any residual lung tissue a mechanical sump pump is recommended—attach to small tube of Bottle B.

with both the inspiratory and compressive phases. Pain, too, both by reflex and by voluntary limitation of costal excursions tends to inhibit deep breathing and free coughing. The author, therefore, never intentionally performs a phrenic paralysis during a pulmonary resection except in those instances where rupture of a tuberculous cavity demands extreme efforts at obliteration of the contaminated pleural space. Phrenic exeresis or phraxis may be performed in a week or two after full respiratory compensation has taken place. Likewise, no more than one rib is removed for entrance into the thorax, and usually none when the anterior approach is used. Careful closure of the pleural rent is always attempted. In order to lessen postoperative pain, the intercostal nerves of the rib above and below the incision are picked up with a hemostat and crushed before closing the chest. Thus, a relatively painless postoperative course is obtained.

Even with the foregoing measures, there are some persons who cannot effectively cough subsequent to these operations. If turning from side to side, sitting the patient up in bed, and the like, prove ineffective, transnasal tracheal suction should be employed by the method perfected by Dr. Lyman Brewer. A relatively new, large-caliber Levine tube is passed through the largest nostril into the pharynx. The tongue is pulled forward and the patient is encouraged to pant vigorously. On an inspiration the tube is passed further, the tip of it tending to enter the glottic aperture. The patient immediately chokes and gasps and is unable to phonate. A strong suction motor is now connected to the Levine tube, which is moved back and forth in the trachea, passing even into the lower lobe bronchi on one or both sides. If the patient becomes cyanotic the suction may be stopped temporarily with the tube in place. The violent bronchial peristalsis induced by this procedure throws much mucus out of the bronchioles into the larger bronchi and trachea, where it is either aspirated or easily coughed up after the tube is removed. This procedure may be performed by the nursing staff at two- to four-hour intervals.

For patients in whom the tube can be passed only with difficulty or where sudden rise of temperature and x-ray evidence of "spread" (really atelectasis first) are observed, formal bronchoscopy should, of course, be done.

In persons requiring frequent tracheal aspirations for copious secretions where there is difficulty in transnasal aspirations, a formal tracheotomy may be done. Then the aspirations may be carried out as often as every fifteen minutes by a catheter inserted through the tracheotomy tube. Between periods of aspiration, oxygen may be administered directly into the trachea by a small catheter passed through the tracheotomy tube. Such oxygen flow should seldom be over 3 L. a minute, and the oxygen should be moistened by bubbling it through water before it reaches the catheter. No difficulty in healing of the tracheotomy sinus has yet been observed.

The patient should be got out of bed as soon as he is able, perhaps by the first postoperative day. This seems to improve both expectoration and the general condition.

Subsequent to an initial successful pulmonary resection, the question of thoracoplasty or phrenic nerve paralysis always comes up. In our series the treatment has varied with increasing experience. While at first no thoracoplasty nor phrenic paralysis was performed except in the treatment of a complication such as an empyema, today one or the other procedure is done as a routine. Thus, a temporary phrenic paralysis is always performed subsequent to a lower lobectomy. A permanent phrenic paralysis and a seven- or eight-rib thoracoplasty are routinely performed after total pneumonectomy. A partial (four- or five-rib) thoracoplasty is performed after an upper lobectomy.

Previously in this paper it has been advised that the pleural space be obliterated quickly and completely by re-expansion of any remaining lung tissue to lessen the risk of tuberculous empyema, and by thoracoplasty where re-expansion obliteration cannot be accomplished. There are two other reasons for reducing the size of the affected hemithorax which to the author seem sound. One is the risk of overexpansion of all remaining lung tissue (on both sides) producing progressive and irreversible destructive emphysema. Much experimental and a growing amount of clinical evidence is available to support this view. The third reason is the presumed risk of breaking down a residual caseous focus within the remaining lung tissue by overstretching this section. Certain of our cases seemed to give presumptive evidence that such breakdown might occur. A huge (giant) cavity developed suddenly in the apex of the residual right lower lobe eleven months after upper and middle lobectomy. This ballooned up so quickly and yet responded so readily to a seven-rib thoracoplasty that the physicians in charge find it hard to believe that the ordinary rules of cavitary formation and closure were applicable in this case. And reasoning backward, if this huge cavity could be controlled after its formation by a seven-rib thoracoplasty, could it not have been more easily prevented by a five-rib operation? (See Fig. 7.)

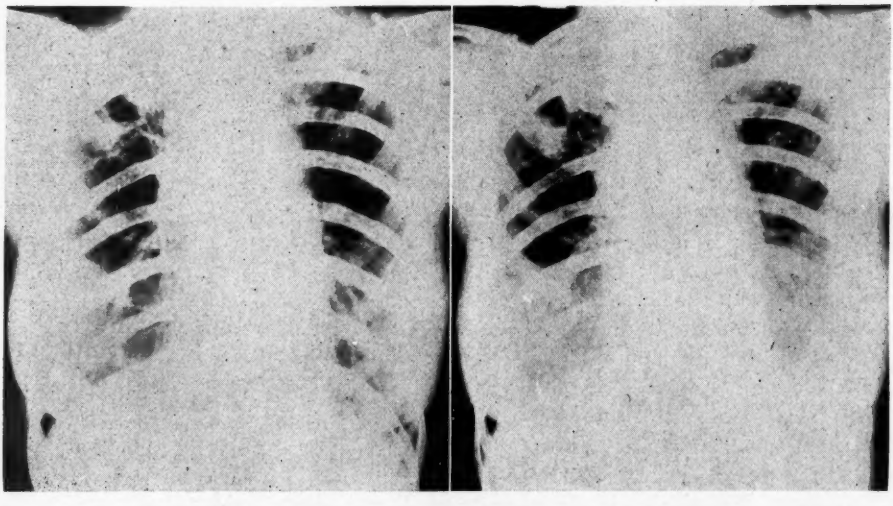
Naturally, in segmental pneumonectomy the lung is only slightly reduced in size, requiring no thoracoplasty.

As to choice of operative procedure, it is obvious that the entire malignant tuberculous focus must be removed if possible; therefore, pneumonectomy will sometimes be necessary. However, if the process is localized in one lobe, it seems unnecessary to remove the other lobes which may contain small nodules. After all, the lobes in the other lung may also contain such nodules, and so may many other organs such as spleen or bones.

Segmental resection has been performed seven times in this series, twice as part of a combined procedure (another lobe being removed at the same time); and five times as an individual procedure. In general, the course of these cases was comparable to that in simple lobectomy. After all, the individual bronchopulmonary segments are functionally as separate as individual lobes, and are not inseparable anatomically.

The indications for this surgery, the procedures performed, and the fatalities are listed in Table I. Four of the cases listed as serious bronchial stenosis were also cases in which thoracoplasty had been previously performed but were not

A. *B.*



C. *D.*

Fig. 7.—*A, B, C, D, E* show development of a large cavity in the overdistended apex of the right lower lobe eleven months after upper and middle lobe lobectomy for tuberculosis. It was readily closed by a seven-rib thoracoplasty. (For Fig. 7, *E*, see opposite page.)

included in the post-thoracoplasty cases. The fatalities and complications listed in Tables I and II are those which have occurred to date. Not all deaths were operative, nor were all due to tuberculosis. An evaluation of the present status of the surviving patients is given in Table III.

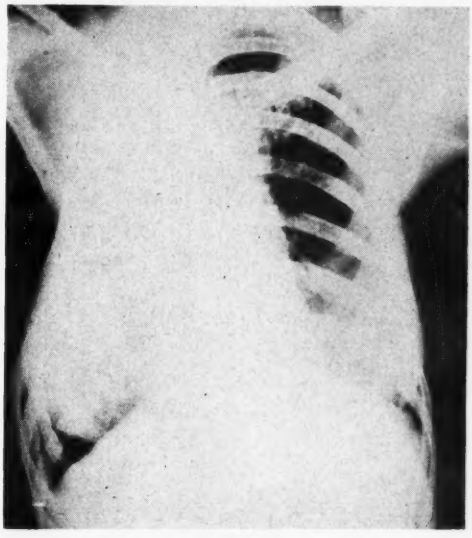


Fig. 7E.

TABLE I

INDICATIONS	CASES	PNEUMONECTOMIES		LOBECTOMIES		SEGMENTAL		% DEAD
		TOTAL	DEAD	TOTAL	DEAD	TOTAL	DEAD	
Serious bronchial stenosis*	9	9	2	–	–	–	–	22.2
Associated or residual bronchiectasis	8	4	0	4	0	–	–	0
Ineffective temporary collapse	4	2	1	2	0	–	–	25.0
Very large cavity	12	5	1	6	2	1	0	25.0
Check valve cavity	3	0	0	2	0	1	0	0
Ineffective permanent collapse	21	9	4	9	2	3	0	28.6
Acute progressive cavitary T.B. (destroyed lungs)	10	9	6	1	1	–	–	70.0
Lower lobe cavity	4	1	0	3	2	–	–	50.0
Elective	7	1	0	5	1	2	0	14.3
Tuberculoma	1	1	1	–	–	–	–	100.0
Total	80	41	15 (36.5%)	32	8 (25%)	7	0	27.5

*Four patients listed as serious bronchial stenosis also had had previous thoracoplasty, but were not so listed.

SUMMARY

Eighty patients with pulmonary tuberculosis have been subjected to pulmonary resection during a four-year period. The indications are the usual ones accepted by surgeons doing pulmonary resection for this disease. The methods

TABLE II. COMPLICATIONS IN THE ENTIRE SERIES TO DATE

COMPLICATION	TOTAL	FATALITIES	PER CENT
Spreads, contralateral	13	6	46.1
Spreads, homolateral	2	0	0
Empyema, tuberculous	5	2	40
Empyema, pyogenic	4	0	0
Empyema, mixed	1	1	100
Bronchopleural fistula plus empyema	7	3	43
Postoperative cardiac failure including pulmonary edema	2	2	100
Operative cardiac arrest, asyphyxial	1	1	100
Due to vagal reflex ?	1	0	0
Chest wall infections and sinuses	6	0	0
Contralateral pleural effusion	2	0	0
Tuberculous meningitis (6 mo. postop.)	1	1	100
Tuberculous pericarditis	1	1	100
Uremia due to amyloid disease (preop.) 3 mo. after operation	1	1	100
Uremia due to transfusion reaction	1	1	100
Acute postoperative anuria	1	0	0
Hemorrhage from a pulmonary vessel (3 days post-tourniquet pneumonectomy)	1	1	100
Slow re-expansion of residual lobes	2	1	0
Late development of bronchopleural fistula	1	0	0
Late development of bronchoesophagopleural fistula	1	1	100
Late re-exacerbation of tuberculosis	2	1	50
Total	56	23	

TABLE III. BREAKDOWN OF PRESENT STATUS OF LIVING PATIENTS

PRESENT STATUS	NO.	%
Satisfactory with negative sputum	39	68.4
Sputum-positive (including the bilateral cases)	6	10.5
Empyema	3	5.2
Draining sinus	5	8.7
Bilateral (preop.)	3	5.2
Spread with progression	3	5.2
Spread without progression	4	7.0

and details of management which have lessened the formidability of the procedure in our hands are given in some detail. The results and complications to date are listed. They seem to indicate in a general way the reasonable expectation of success which may be entertained in the various types of pulmonary tuberculosis.

CONCLUSIONS

Pulmonary resection for tuberculosis is a practical method of removing pathologic foci, which resist collapse measures. The actual morbidity associated with these procedures cannot be accurately estimated at the present time, since our earliest cases were operated on and managed by antiquated and deficient methods, and since very few cases were operated on even five years ago (none in this series over four years ago and many were operated on recently). Since operative complications may not cause death for months or years, the actual operative mortality is difficult to estimate. Statistics of different clinics vary considerably because some physicians may call a contralateral flare-up a spread, while others call it autotuberculinization. Furthermore, one series may include

a considerable proportion of patients with a very poor prognosis, such as acute caseous pneumonia, while another may be largely composed of patients with a good prognosis, such as bronchial stenosis or residual bronchiectasis. In still another series they may be largely of post-thoracoplasty patients. Here the operative trauma and shock may be exaggerated by the distorted anatomy, and a considerable risk of operative cavitary rupture is ever present.

But when all this is allowed for, it must be accepted that pulmonary resection is applicable in many cases where no other method of treatment is feasible, and in such patients it presents an operative mortality of probably less than 20 per cent. Further efforts and the elapse of several years will be necessary before exact evaluation is possible.

INTRABRONCHIAL HEMORRHAGE IN BATTLE CASUALTIES

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INTRODUCTION

IN THE Sicilian and Western European Campaigns, 118 men having thoracic wounds were treated by the writer and other members of an auxiliary surgical team. The group included thirty-three with thoracoabdominal wounds and eighty-five with penetrating or perforating thoracic wounds (Table I). Most of these were the more severe "nontransportable" casualties as seen in Field Hospitals. A clinical triad, intrabronchial hemorrhage, ineffectual cough, and anoxia, was of frequent occurrence. In these patients, prompt relief of the bronchial obstruction was urgently required. This report considers the etiological factors, the clinical features, and the method of treatment that was evolved in the management of the respiratory embarrassment in this group. Emphasis is placed on the necessity for immediate and thorough clearing of the tracheobronchial tree as a part of resuscitation. The advantages of bronchoscopic aspiration to accomplish this objective is stressed. A technique of instrumentation without anesthesia, with simultaneous administration of oxygen, and without moving the patient from a semisitting position is advocated. The method proposed was generally used by anesthetists and surgeons of the Third Auxiliary Surgical Group, the members of which performed over 19,000 operations under field conditions in Western Europe, among which 1,306 bronchoscopic aspirations are recorded.

The intrabronchial hemorrhage in the patients to be described was never severe. Impairment of cough was always present, however, and most of the patients having severe symptoms had not been able effectively to clear their air passages for a period of from six to twelve hours. Invariably, those presenting the more severe degrees of anoxia were those in which ineffectual cough had resulted in a "spill" of blood and secretions into the unaffected lung. The factors that were noted to impair cough were, in order of their importance:

1. Dyspnea due to hemothorax and bronchial obstruction
2. Pain and the fear of pain caused by coughing
3. Shock
4. Sucking wounds
5. Lacerations of the diaphragm
6. Cerebral anoxia
7. Morphine narcosis.

It seems likely that in past wars those sustaining thoracic wounds productive of severe respiratory embarrassment have either been included among

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TABLE I. THORACIC WOUNDS

TYPE	NUMBER	NO. DEATHS	% MORTALITY
Penetrating	35	1	2.8
Sucking	50	7	14
Thoracoabdominal	33	6	18.1
Totals	118	14	11.8

the high percentage of chest wounds that are fatal on the battlefield[1] or the men have not survived to reach a hospital. The frequency with which casualties of this type were seen in the recent conflict was doubtless due, in part, to the proximity of hospitals to the front. No less important, however, was the prompt treatment received for sucking wounds and shock in aid stations and the reduction in the time required for evacuation.

BIBLIOGRAPHY

For reasons considered above, the subject under consideration has received scant attention in all except the most recent literature of military surgery.

Jolly[2] writing of his experience with chest wounds in the Spanish Civil War, states: "Almost 90 per cent of the total number of cases show hemoptysis, but in those in which the wound was caused by a bullet, the bleeding rapidly diminishes and usually ceases towards the end of the first week." Again, speaking of the operative treatment for laceration of the lung, he says: "Hemoptysis is seldom severe enough to cause alarm. It ceases spontaneously under general treatment, especially repeated injections of morphine." It can be inferred that these patients had comparatively minor wounds and that their cough mechanism was effective. As will be shown later, the operative difficulties encountered in the thoracic operations reported here, were, in the main, those connected with keeping the airways clear of blood. That Jolly experienced the same difficulty can be deduced from his account of the surgical problem involved in the repair of the lacerations of the lung and diaphragm. Regarding this, he says: "The operation must be performed quickly . . . the patient's condition is precarious throughout . . . many of the more gravely wounded men die on the table."

In Bailey's more recent and more comprehensive volume concerned with war surgery, Edwards[3] does not consider this subject. Magill[4] in the same volume, writing on the anesthesia in thoracic injuries considers the problem briefly: "It is essential to maintain a free airway at all times . . . intubation should be reserved for those operations during the course of which it may be necessary to aspirate blood from the trachea and bronchi." He does not suggest bronchoscopy.

Churchill,[5] writing on his observations of chest injuries in the Mediterranean Theater, makes specific but brief mention of the difficulties caused by bloody secretions in the airways, as follows: "The use of bronchoscopic and catheter tracheobronchial aspiration to clear the respiratory passages of bloody secretions has found increasing use." His observation that oxygen therapy is ineffectual without such aspiration has been confirmed many times in this study.

Sampson[6] working in the same theater, appears to be more enthusiastic in the application of bronchoscopy. In a recent letter he wrote: "We agree perfectly about the value of aspiration of the bronchial tree whenever or wherever there is difficulty in raising. As a teaching principle for other teams we have probably done more catheter aspirations, since this is a bit easier to teach. However, there is no doubt that bronchoscopy is more efficient than the catheter. We have been surprised that bronchoscopy can be repeated so often without ill effect."

In more recent communications regarding general hospital experience with thoracic wounds[7, 8] tracheobronchial aspiration is either ignored or given only passing consideration. In general, it may be noted that the application of tracheobronchial aspiration is advocated most warmly by those whose experience was gained with "nontransportable casualties" in field hospitals. In this connection, comparison of the article of Betts[9] with that of Welch and Tuhy[10] on thoracoabdominal wounds is of interest. The former's experience was largely in field hospitals and was presumably similar in every way to that reported here. He strongly advocates tracheobronchial aspiration postoperatively and states: "We believe that all members of any thoracic surgical team, surgeon, assistant surgeon, and anesthetists, should be competent bronchoscopists in order that the benefits of bronchoscopy will always be available." Welch and Tuhy's experience was in an evacuation hospital. They consider tracheobronchial aspiration in neither pre- nor postoperative treatment. Among other postoperative pulmonary complications they record five instances of atelectasis and four of bronchopneumonia in seventy operative cases, an incidence of 12.8 per cent. Betts,* in an experience with thirty-one field hospital patients who were presumably more gravely wounded, records two fatal pulmonary complications, one from "strangulation," and another from lobar pneumonia, an incidence of 6.4 per cent, or exactly half that of the former series.

In a communication from the Mediterranean Theater, Snyder[11] considers the preoperative role of tracheobronchial aspiration in chest wounds. He advocates intercostal nerve block and catheter aspiration, but adds that, "in some instances bronchoscopic aspirations must be used." He recommends routine aspiration through the bronchoscope at the end of the operation.

Beecher[12] in an article of anesthesia practice, from the same theater, opposes the routine use of the bronchoscope in thoracic injuries, but advocates tracheobronchial aspiration. His objection to bronchoscopy at the end of operation because it necessitates deep anesthesia or the application of a local anesthetic is not substantiated in this study. As will be shown later, bronchoscopy can be safely done in these patients without any anesthesia, local or general, and for various reasons omission of any anesthetic is advantageous.

In contrast with the foregoing, specific and detailed consideration of this subject is found in two more recent communications from the Mediterranean Theater which consider casualties of this type under the title of "Wet Lung." In the first, Burford and Burbank[13] report seventy-nine patients "first seen within forty-eight hours after being wounded, the majority of which were seen

*In a personal communication he reports one nonfatal case of atelectasis.

within twenty-four hours." They postulate the view that "in all wounds of the chest, to a greater or lesser degree, depending upon the type and severity of the lesion, the lung tissue reacts to produce more than its usual amount of interstitial and intraalveolar fluid." They suggest the "existence of reflex bronchial and bronchiolar spasm originating from a painful chest wall or parietal pleura, which would explain some of the phenomena observed." In their treatment, they stress the desirability of "abolition of pain and physiologic remobilization of the chest wall" by intercostal injections (novocain nerve block). With this treatment they report that "the cough becomes painless and effective, and evacuation of the bronchopulmonary tree becomes established." In some instances they utilized tracheobronchial catheter aspiration, and state that, "in a few, bronchoscopic aspiration may be necessary."

The second communication of Brewer and his associates[14] considered under the heading of "Causative Factors": "Forces leading to the production of secretions and other fluids in abnormal amounts in the respiratory tract" and "conditions preventing adequate removal of the fluids so produced." To support the view that abnormal amounts of secretions are produced, they cite the experimental work of Drinker and Warren[15] and make clinical application of the findings of these authors. They do not evaluate the relative importance of these factors. Like Burford and Burbank, they advocate bronchoscopy only after intercostal nerve block or catheter suction has been tried. They state that topical anesthesia for bronchoscopy must be employed "in all except semicomatose or moribund patients." In addition, they advise that 100 per cent oxygen should be administered through the bronchoscope during this procedure to all patients who have anoxia. These practical features of treatment will be discussed later.

The observations of these authors that patients in this category may be confused with pulmonary edema has been confirmed in this study. The familiar clinical signs of the latter, together with the frothy, bloody sputum should make the differential diagnosis easy. In the group reported here, pulmonary edema was occasionally seen in patients who had been given large amounts of plasma prior to whole blood transfusion. They were commonly in a state of irreversible shock.

The writer's experience indicates that, in general, ineffectual cough alone is the basis for the symptoms in this type of injury, and that the benefits reported by these authors with novocain injections are due entirely to the increased effectiveness of cough which followed relief of pain. This view is supported by our observation that striking relief which persisted for several hours usually followed thorough bronchoscopic aspiration (Cases 1, 2 and 3). Pulmonary edema excepted, persistence of "wetness" after any treatment short of bronchoscopy, performed as hereinafter described, appears to the writer to be due to incomplete clearing of the tracheobronchial tree. In general, we preferred to use what is admittedly the most effectual method of clearing the airways (bronchoscopy) at once, correct shock, and then proceed, in most instances, with a definitive operation performed under endotracheal anesthesia, at which

time ribs were trimmed and lacerations of the chest wall, lung, and diaphragm were repaired. Bronchoscopy was done routinely at the end of operations and, subsequently, cough was usually comparatively painless and effectual.

ETIOLOGICAL CONSIDERATIONS

The wounds producing the clinical triad previously noted varied greatly. They were rarely caused by small-arms fire. More commonly the injury was produced by a high explosive shell fragment. Usually the foreign body was 1 cm. or more in diameter. It was retained in some instances, and in others the wound was of the "through and through" variety. One, and occasionally two, sucking wounds were usually present and single or multiple shattered ribs were the rule. A fairly extensive laceration of the lung was usually present. Laceration of the diaphragm was common. Hemothorax was always observed; hemopneumothorax was somewhat less frequent. A sizeable bronchial fistula permitting a leak of the hemothorax blood into the bronchi was exceptional. Wounds of the latter type doubtless commonly occurred, but those sustaining them probably died before reaching a hospital. The source of the intrabronchial blood, with one exception, appeared to be a slow pulmonary hemorrhage into a small bronchus.

The following observations support this view:

1. Most of the patients survived six to twelve hours after being wounded, during which time they had not coughed effectively.
2. Active bleeding was not found at bronchoscopy in any instance.
3. The relief that followed aspiration usually lasted several hours.
4. Instrumentation was never observed to increase hemorrhage.

CLINICAL FEATURES

Serious intrabronchial hemorrhage does not present a diagnostic problem. In severe instances there were audible tracheal râles, orthopnea, and cyanosis. In addition, disorientation, excitement, and restlessness, the results of cerebral anoxia, were commonly present. The physical and x-ray examination revealed signs of the hemothorax or hemopneumothorax which was invariably present. Subcutaneous emphysema was frequent. More important was the presence of palpable râles. The latter is a sign of great value. In penetrating chest wounds this may rarely be due to pulmonary edema but for practical purposes it was diagnostic of retained bloody secretions in the tracheobronchial tree, causing serious bronchial obstruction which can be most effectively relieved by bronchoscopy. It also offers a simple and rapid method of determining the affected lung, and roughly delineating the lobes involved. As previously noted, the most severe anoxia was seen in those in whom there had been a "spill" of bloody secretions into the unaffected lung.

TREATMENT

Bronchial hemorrhage producing anoxia of the gravity described requires prompt treatment. Clearing the tracheobronchial tree takes precedence over all other resuscitative and diagnostic measures. Aspiration of the secretions

should be done regardless of the patient's condition. Suction through an intratracheal catheter is valuable to accomplish this objective. Greatly to be preferred, however, is bronchoscopic aspiration which permits visualization of the branch bronchi of the individual lobes. Experience reported here permits this assertion to be made categorically. Secretions producing "palpable râles" over a bronchopulmonary segment, which could not be aspirated through an intratracheal catheter, have been repeatedly removed through the bronchoscope.

The patient's condition may be precarious when he is first seen. His head and shoulders have usually already been elevated, if such is the case, and oxygen is being administered. It is not safe to lower the head of such patients or even temporarily stop the administration of oxygen. These men have been bronchoscoped on army cots in approximately the position in which they lay. It is possible to visualize the main bronchi readily in this position by having an assistant lift the chest slightly (Fig. 1). Oxygen administration is started by the nasal catheter method before passage of the instrument and preparation is made to change the flow to the sidearm of the bronchoscope immediately after passing the glottis. Failure to observe this precaution may result in a fatality during instrumentation. Aspiration usually yields 30 to 60 c.c. of blood and mucus secretion. The chest should be palpated again after clearing the trachea and the main bronchi. "Palpable râles" frequently remain over one or more lobes depite aspiration of large amounts of secretion. It is then advantageous to apply the aspirator tip directly to the orifice of the affected lobe. This excites cough and usually the secretions are expelled and immediately withdrawn. No râles can be felt at the completion of a properly conducted bronchoscopy.

Subsequent management required definitive treatment for the associated injuries. In the exceptional patient there was only a minor wound of the chest wall and lung associated with a hemothorax. Debridement of the former and aspiration of the latter may be the only treatment required in injuries of this type. More commonly, there were associated sucking wounds, lacerations of the lungs, tears of the diaphragm, or a thoracoabdominal wound. Tension pneumothorax was only occasionally observed. The above wounds required appropriate surgical treatment which will not be discussed here. It was found that it was usually preferable to operate soon after clearing the air passages, provided a satisfactory response was made and shock was not present.

It was necessary occasionally to do another bronchoscopic aspiration after anesthesia had been induced before introducing an intratracheal catheter. Bronchoscopic aspiration at the end of operations was done routinely. These patients, if encouraged and assisted, were usually able to cough after operation. Occasionally this was not the case and in these, prompt bronchoscopic aspiration was repeated as often as necessary until the bleeding stopped or effective cough was re-established.

TECHNIQUE

Battle conditions require improvisation. The first endoscopies were done without trained assistants or anesthesia. As already noted, many patients were on army cots, oxygen was being administered, and the patient's condition

was of such gravity that he could not be moved. It was found that these patients should be "bronchoscoped where they lay." Under these circumstances it was learned that satisfactory endoscopy could be done in unconventional positions and that anesthesia was unnecessary. It is now felt that for the following reasons, anesthesia is not only unnecessary, but actually undesirable:

1. Valuable time is consumed which might otherwise be used in operating upon other gravely wounded patients.
2. Efforts to apply an anesthetic may produce ineffectual cough. This exposes the patient to the danger of displacement of secretions into the remaining functioning lung with disastrous results.
3. Active cough reflex during instrumentation preserves the "tussive squeeze"[16] and greatly assists in emptying the small bronchi.
4. Even prolonged efforts to produce anesthesia are comparatively ineffectual because of excessive amounts of secretion in the airways.
5. No instance of failure to pass the bronchoscope, or injury to the patient was experienced, despite occasional complete lack of cooperation in robust and disoriented patients.

Fig. 1.—Position used for bronchoscopic aspiration in seriously anoxic patients. Note that oxygen is being administered through the bronchoscope.

Reference has already been made to an unconventional position used in bronchoscoping patients on a cot. More commonly, aspiration was done on an improvised operating table made with a litter. Here it was advantageous merely to elevate the patient's head in introducing the instrument (Fig. 2). Visual-

ization of the lower bronchi necessitated raising the patient's chest slightly since the head could not be lowered. Flagg's laryngoscope, which eliminates one wire connection, was found preferable.

MORTALITY

Indications among combat casualties for active measures to remove blood and secretions from the tracheobronchial tree arose invariably in patients presenting lacerations of the lung and sucking wounds. Three of the seven operative deaths that occurred in the group were directly or indirectly attributable to intrabronchial bleeding. One patient who had a massive hemorrhage into the bronchi during operation will be described (Case 4). The others had severe and persistent hemoptysis after the operation. Cough was fairly effectual but postoperative pneumonia developed and death occurred on the

Fig. 2.—Position of patient after bronchoscope is in place for aspiration of lower bronchi on an improvised operating table.

seventh and eighth postoperative day. A fourth patient died suddenly twenty-four hours after operation, presumably from tension pneumothorax. He was operated upon during a period before closed intercostal subaqueous drainage was routinely established at operation; of the remaining three, one died of shock during the operation, a second whose shock was never corrected before

operation died from irreversible shock twenty-four hours after thoracotomy. The third patient died suddenly twenty-four hours after operation from unknown causes. The one death that occurred in the group having penetrating wounds occurred during induction of anesthesia. An autopsy was not done.

Of the six fatalities occurring in the thoracoabdominal group, one died from pneumonia, one from massive hemorrhage from a lacerated liver, three from shock and anuria, one from air embolism due to an associated neck wound.

CASE REPORTS

CASE 1.—The following patient was treated in a field hospital in the early days of the Sicilian Campaign and is believed to be the first battle casualty to have received emergency bronchoscopic aspiration in the field. He is an example in which bronchoscopic aspiration constituted practically the only definitive treatment necessary.

A 23-year-old patient sustained a penetrating, high explosive shell fragment wound of his right chest, with resultant hemopneumothorax and intrabronchial bleeding. He was dyspneic, cyanotic, and disorientated on admission. Bubbling tracheal râles and palpable râles were present over both lungs. Exploration with a large gauge needle disclosed a shallow pneumothorax from which air escaped under pressure for a few minutes. He was not relieved. Withdrawal of 700 c.c. of blood from the right pleural sac a short time later was followed by very little improvement. Anoxia was extreme seven hours after admission. Bronchoscopy with aspiration of about 60 c.c. of bloody secretions gave immediate relief. His color became pink during the procedure and following it his respirations were quiet and the râles disappeared. His condition remained excellent for twelve hours. Increased bleeding into the bronchi then led to almost identical symptoms. Another bronchoscopic aspiration again gave relief. At this time the wounds were debrided. This patient was evacuated five days later and is known to have arrived at a distant hospital in good condition.

CASE 2.—This patient presented essentially the same problem as the one described above. The wounds in both were comparatively minor. More effectual cough after bronchoscopy prevented accumulation of bloody secretions for a longer period.

A penetrating, high explosive shell fragment wound of the right chest produced hemothorax and intrabronchial hemorrhage. Cyanosis, dyspnea, and palpable râles were present. Bronchoscopic aspiration of about 75 c.c. of bloody secretions gave immediate relief. Oxygen by B.L.B. mask was administered for the next twenty-four hours and the patient was able to raise a small amount of bloody secretion. Symptoms then recurred and a second bronchoscopic aspiration was necessary. Subsequently, his condition improved steadily. Aspiration of the hemothorax and debridement of the wound of entry was the only additional treatment necessary. He was evacuated seven days later in good condition.

CASE 3.—This patient illustrates the problem presented in instances of severe sucking wounds with shattered ribs and lacerated lung.

A patient, aged 26 years, was wounded by a high explosive shell fragment which entered through the right shoulder, fractured the scapula, and shattered the underlying fourth and fifth ribs. It then made its exist by avulsing the spine of the adjacent vertebrae and dividing most of both the rector spinae muscle groups. Hemopneumothorax, intrabronchial hemorrhage, and tension pneumothorax were present on admission. Deflation of the pneumothorax and bronchoscopic aspiration were followed by operation performed under intratracheal ether-oxygen anesthesia. Another bronchoscopic aspiration was necessary at the end of the operation. The patient made a slow recovery from the anesthesia and four hours later excessive bloody secretions were again causing grave respiratory embarrassment. A third bronchoscopic aspiration was done, following which he was able to raise effectively a large amount of bloody sputum which continued for seventy-two hours. He was evacuated eight days after operation in good condition.

CASE 4.—This patient demonstrates the danger attendant to operation when a large bronchial fistula is present. Death occurred on the operating table due to flooding of the bronchi with blood. The source of the latter appeared unquestionably to be the hemothorax.

This patient presented a sucking wound of the right chest. The shell fragment entered posteriorly near the inner border of the lower end of the scapula. It shattered two ribs, penetrated both lower and upper lobes of the right lung, and lodged superficially in the latter at the level of the second rib. He was expectorating moderately large amounts of bloody sputum and was not in respiratory distress. During induction of ether-oxygen anesthesia, a fairly large amount of bloody secretion accumulated in the tracheobronchial tree. Aspiration of the latter through a bronchoscope was done before intubation. A few minutes after the operation was begun, there was a sudden, massive flooding of the tracheobronchial tree with blood. Death followed immediately. Examination of the affected lung showed an extensive laceration of the upper lobe and division of a large branch bronchus.

COMMENT

The application of suction bronchoscopy to field surgery is by no means limited to the group of patients described. In addition, this treatment was used in several instances of asphyxia from aspiration of blood or vomitus. During the latter part of the war such aspirations were used almost as a routine in a larger group undergoing extensive abdominal operations. It is of interest to note that in the abdominal group there was none, and in the thoracic group there was only one instance of postoperative pulmonary atelectasis.

The advisability of using aspiration through an intratracheal catheter rather than bronchoscopic aspiration is admittedly debatable. That bronchoscopic aspiration is more effectual cannot be questioned. Experienced bronchoscopists in numbers sufficient to perform this work, however, are not available. The writer's anesthetist, who was skilled in intratracheal intubation, readily acquired a working knowledge and skill in bronchoscopic aspiration. In the latter part of the war he* performed most of the endoscopies necessary in this series.

Interest in and application of bronchoscopic aspiration in the patients under consideration became general among the surgeons and anesthetists in the Third Auxiliary Surgical Group. Despite the fact that in most instances they were previously inexperienced, one member at least of practically every team† acquired a technique adequate to cope with these emergencies. It is the writer's conviction that the application of suction bronchoscopy is a skill that can and should be readily acquired by professional anesthetists. The acquisition of this technique by those working in a combat zone is particularly desirable.

SUMMARY

1. A clinical triad, intrabronchial hemorrhage, ineffectual cough, and anoxia commonly occurring in casualties having "nontransportable" thoracic wounds, has been described. The source of the intrabronchial hemorrhage and the factors which impair the cough mechanism have been noted; the clinical picture resulting has been described in appropriate treatment outlined.

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†Four officers and four enlisted men.

2. The experience recorded indicates that cases of this type occur frequently in hospitals operating near the front when proper first aid is applied and the casualties are promptly evacuated.

3. It has been shown that in some patients aspiration of the tracheobronchial tree is practically the only definitive treatment necessary. It constitutes an essential part of the resuscitative treatment and anesthesia in those who are more severely wounded.

4. Two techniques of bronchoscopic aspiration applicable to surgery in the field have been described; the advantages of instrumentation without anesthesia have been considered. It is emphasized that the gravity of the patient's condition does not contraindicate such treatment. Indeed, the more grave his condition, the more urgent is the necessity for such aspiration.

5. Bronchoscopic aspiration has never been observed to increase intrabronchial bleeding and repeated endoscopies are tolerated without ill effect.

REFERENCES

1. Carter, Noland B., and DeBakey, Michael E.: Current Observations on War Wounds of the Chest, J. THORACIC SURG. **13**: 272, 1944.
2. Jolly, D. W.: Field Surgery in Total War, New York, 1941, p. 158. Paul B. Hoeber, Inc.
3. Edwards, A. Tudor: Surgery of Modern Warfare, Bailey, Hamilton, Baltimore, 1942, Williams & Wilkins Co., vol. 1, chap. 25, pages 369-388.
4. Magill, I. W.: Surgery of Modern Warfare, Bailey, Hamilton, Baltimore, 1942, Williams & Wilkins Co., vol. 1, chap. 36, page 390.
5. Churchill, E. D.: Trends and Practices in Thoracic Surgery in the Mediterranean Theatre, J. THORACIC SURG. **13**: 308, 1944.
6. Sampson, Paul C.: Personal communication.
7. Johnson, Julian: Battle Wounds of the Thoracic Cavity, Ann. Surg. **123**: 321, 1946.
8. Burke, John, and Jacobs, Theodore T.: Penetrating Wounds of the Chest, Ann. Surg. **123**: 363, 1946.
9. Betts, Reeve H.: Thoracoabdominal Injuries, Ann. Surg. **122**: 793, 1945.
10. Welch, C. S., and Tuhy, J. E.: Combined Injuries of the Thorax and Abdomen, Ann. Surg. **122**: 358, 1945.
11. Snyder, Howard E.: The Management of Intrathoracic and Thoracoabdominal Wounds in the Combat Zone, Ann. Surg. **122**: 333, 1945.
12. Beecher, Henry K.: Anesthesia for Men Wounded in Battle, Ann. Surg. **122**: 807, 1945.
13. Burford, Thomas H., and Burbank, Benjamin: Traumatic Wet Lung, J. THORACIC SURG. **14**: 415, 1945.
14. Brewer, L. A., Burbank, Benjamin, Sampson, P. C., Schiff, C. C.: The "Wet Lung" in War Casualties, Ann. Surg. **123**: 343, 1946.
15. Drinker, Cecil K., and Warren, Madeleine F.: The Genesis and Resolution of Pulmonary Transudates and Exudates, J. A. M. A. **122**: 269, 1943, cited by Brewer et al.
16. Jackson, Chevalier, and Jackson, Chevalier L.: Bronchoscopy, Esophagoscopy and Gastrostomy, Philadelphia, 1934, W. B. Saunders Co., chap. 11, page 66.

THORACOSCOPY AND PNEUMONOLYSIS IN PULMONARY TUBERCULOSIS

NOTES ON TECHNIQUE AND IMMEDIATE RESULTS

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THE following study is based upon the experience gained in 500 cases, with 654 operations performed, during the period of 1934 to 1945 by several thoracic surgeons at the Grace Dart Home Hospital, the Royal Edward Laurentian Hospital, and three general hospitals in Montreal. It includes operations on Army and ex-Army personnel carried out by the author in 1943 and 1944 under arrangement with the Department of Veterans Affairs. An additional number (100 operations in seventy-six cases) was performed since the preparation of this paper and is not considered in the postoperative statistics.

It is not intended to give a complete analysis of the cases with a follow-up study of the clinical results. This task has to be postponed until a sufficient period of time has elapsed to allow for an accurate account of the end results. Many details about instruments, technique, etc. have to be omitted because of lack of space. It is the writer's intention, however, to take a definite stand regarding the major problems in technique and with reference to the important and difficult task of choosing the right cases and the right adhesions for this operation.

INSTRUMENTS

During the first five years of our series of operations we used the instruments designed by Jacobaeus, with the modifications of Unverricht. In later years we changed over to the Coryllos thoracoscope. When high frequency electrodes were advisable we used the unit made by the same manufacturer (A.C.M.I.). In addition to these instruments, a cautery blade bent into the shape of a hook and a suction tube are occasionally very useful.

ADHESIONS

Table I shows the different types of adhesions that were encountered in our cases. Adhesions containing blood vessels were not considered separately. Blood vessels prevented a complete separation in only a few earlier cases (0.8 per cent). The type of adhesion is brought into relation with the operative procedure and it is clearly shown how the success of pneumonolysis diminishes with the more difficult type of adhesions. It is necessary to define the terms used in the table. *Complete pneumonolysis* means that all visible adhesions were severed. *Partial pneumonolysis* means that either some adhesions were

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cut completely and others left alone on account of difficulties which were encountered, or that an extensive adhesion, usually a web adhesion in the posterior mediastinum or a spool adhesion at the apex, were cut partially to the extreme point of apparent safety. *Thoracoscopy* means that the visualized adhesion was not suitable for cutting or that no adhesions were found.

Solitary or multiple string and band adhesions were usually found between lateral, anterior, or posterior chest wall and the convexity of the lobes (upper lobe most frequently). We encountered spool adhesions chiefly between the apex of the upper lobe and the pleural cupola (first rib and subclavian vessels). Web adhesions and shelves were more numerous posteriorly in the mediastinal gutter. Adhesions to the diaphragm were usually string- or bandlike in character and ran from the outer lower border of the lung to the costophrenic angle. Diffuse adhesions from the base of the lung to the diaphragm were presumed by their x-ray appearance and when the lung could not be lifted by means of a blunt ribbon retractor.

TECHNIQUE OF PNEUMONOLYSIS

Anesthesia.—All patients received ¼ grain morphine and 1/100 grain scopolamine one-half hour before operation. We prefer local anesthesia. A general anesthetic (N_2O) was used only in a very few cases and did not give encouraging results.

Introduction of Instruments.—Most adhesions are situated in the posterior or axillary region. The lung, therefore, collapses in a rather anteroposterior direction and allows sufficient air space in the anterior chest. Posteroanterior x-rays may show only a very small zone of pneumothorax, while oblique x-rays, tomographic or stereoscopic views, may indicate better the most suitable place for the introduction of the trocar.

We introduce the scope according to the individual case. In 488 instances out of 754 stages the first puncture was made in the anterior axillary line at the fifth interspace. In the remainder, the trocar was inserted anywhere between midclavicular and midscapular line.

The inserted scope indicates where the cannula for the cautery should be introduced. In the great majority of operations, the fifth to the seventh interspace in the posterior axillary line was chosen.

In the posterior half of the chest, the intercostal vessels and nerves are protected by the lower rib margin. If, therefore, the trocar be inserted in an oblique direction from above downward, there is little likelihood of damage. Anterior vessels and nerves are exposed, but they can be easily pushed away as the spaces between the ribs are widened.

Almost all postoperative hemorrhages into the chest cavity are due to bleeding from the trocar opening (intercostal vessels) and rarely from a severed adhesion. The wall of the trocar exercises a temporary hemostasis while withdrawal of the instrument invites new bleeding. We have observed this event on several occasions through the thoracoscope.

TABLE I. TYPE OF ADHESION IN RELATION TO OPERATIVE PROCEDURE IN 576 OPERATIONS (TWENTY-ONE BILATERAL)

TYPE OF ADHESION	OPERATIVE PROCEDURE									TOTAL	
	COMPLETE PNEUMONOLYSIS			PARTIAL PNEUMONOLYSIS			THORACOSCOPY				
	CASES	% ADHESIONS	% PROCEDURE	CASES	% ADHESIONS	% PROCEDURE	CASES	% ADHESIONS	% PROCEDURE	CASES	PER CENT
String, band, and cord adhesions	141	60.0	88.7	17	5.6	10.7	1	1.8	0.6	159	26.6
Same, combined with web and shelf adhesions (not mediastinal)	66	28.1	42.0	86	28.1	54.8	5	8.9	3.2	157	26.3
Solitary or combined adhesions, the majority of which are:											
Spool adhesions	18	7.7	15.8	76	24.8	66.7	20	35.7	17.5	114	19.1
Mediastinal adhesions	2	0.8	2.2	79	25.8	87.8	9	16.2	10.0	90	15.1
Diaphragmatic adhesions	0	0.0	0.0	13	4.3	100.0	0	0.0	0.0	13	2.2
Diffuse adhesions or partial synechia of lung to chest wall	8	3.4	13.6	35	11.4	59.2	16	28.5	27.2	59	9.9
No adhesions found	0	0.0	0.0	0	0.0	0.0	5	8.9	100.0	5	0.8
Total	235	39.4		306	51.2		56	9.4		597	100.0

Evaluation of Adhesions.—After aspiration of the fluid, if such be present, inspection of the adhesions is necessary. Their position should be carefully checked with the last available x-ray. An adhesion still visible in the x-ray may have ruptured before the operation (5 cases). Very often, adhesions to healthy lobes are rather useful as they allow a selective collapse of the diseased parts after division of the adhesions to the affected lobes. Selective collapse is especially important in those cases where any collapse therapy on the contralateral side is contemplated or already performed. The presence of a peripheral cavity in the x-ray demands special precautions. The possibility of a tension cavity should be kept in mind, since separation of the adhesion may be followed by rupture of the cavity into the pleural space.

Separation of Adhesions.—Adhesions which may contain lung tissue are usually divided by decollement of the parietal pleura. Occasionally this procedure may be assisted by the hydraulic pressure of injected fluid (novocaine or saline) into the plane of cleavage. For thick adhesions which cannot be transilluminated, or mediastinal adhesions, the hook cautery is very useful. It picks up small layers and cuts them separately. In small string adhesions, distant from the optic and dangerously near to the great vessels, "hooking" serves the same purpose.

Special attention should be given to blood vessels within the adhesion. Within the last 10 years, the galvanocautery became a stepchild while the high

frequency cautery (Matson) gained more and more popularity. A word in favor of the galvanocautery may be said.

A cautery blade, red hot and used at its edge, will deposit a great amount of heat at one very small spot. The tissue, especially in a string or band adhesion, will be charred in the immediate neighborhood, but the dried parts will prevent the further conduction of heat to remote parts of the adhesion.

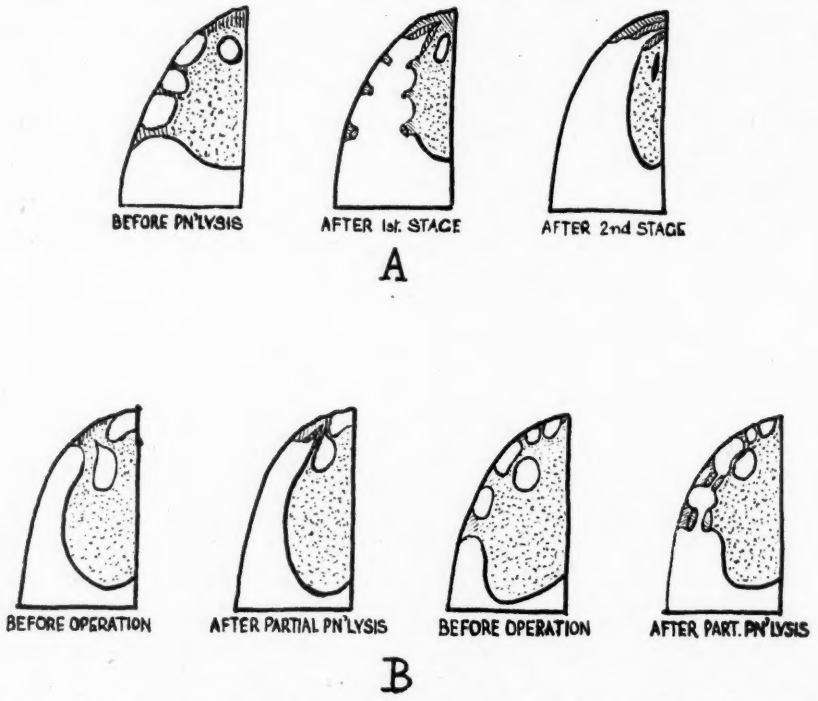

Fig. 1.—Partial pneumonolysis. *A*, pneumothorax should be continued and a further stage attempted. *B*, pneumothorax should be abandoned immediately.

If there is a blood vessel within the adhesion, it will be severed, but the very narrow zone of charred tissue will rather prevent a contraction of the vessel wall and the small clot is not firm enough to stop the blood flow. Bleeding can be expected even hours after the operation. This method, therefore, is not recommended.

The application of the cautery with its flat side to the adhesion and dull red (visible only in the dark) will heat up the adhesion slowly and uniformly. It will cause coagulation of a larger field, even if the adhesion contains a blood vessel of considerable size. The edge of the cautery will readily cut through the coagulated zone while the blood vessels are well secured by coagulation and subsequent shrinking of the surrounding tissue. The high frequency

cautery has its place at special occasions but it is by no means the key to a successful separation of blood vessels.

In many instances, adhesions can be severed only partially, due to their shortness or the lack of a free edge to work on, or they are so diffuse that the operation extends over several hours and the patient becomes uncomfortable. The question arises whether to abandon any further attempt or to try separating again at a later stage. No fast rules can be given. If the remaining adhesion is near a peripheral cavity, it is far better to abandon the pneumothorax immediately (Fig. 1, *A* and *B*). If there is no cavity in the neighborhood of the adhesion and the visceral pleura appears healthy, the pneumothorax could be continued and a further stage of pneumonolysis attempted.

Closure of Trocar Opening.—Intrapleural pressures should be adjusted before or immediately after closure of the trocar openings and should be on the negative side. The closure of the wound itself is usually carried out by one or two sutures or clips. This type of closure, however, is far from ideal as it does not include the parietal pleura. No air will escape or enter between the outside and the thoracic cavity, but air may enter into the fascial planes from the inside and cause a subcutaneous emphysema. Its extent depends on the intrapleural pressures and the size and number of trocar openings. None of the methods of closure described in the literature have been successful in the prevention of subcutaneous emphysema in our experience. Recently, we tried the high frequency current for coagulation of the wall of the trocar opening as described by Vineberg and Kunstler for the intracavitary suction drainage. We believe that this method may reduce the incidence of emphysema and hemorrhage after withdrawal of the instruments.

Postoperative Care.—Special attention is paid to the fever chart. Failure of fever to subside after a week or a rise of temperature forty-eight hours after the operation is considered with suspicion. Routinely inspiratory and expiratory x-rays are made the next day in spite of the sometimes difficult interpretation due to subcutaneous emphysema. Refills are accomplished depending upon the information thus obtained.

AGE AND SEX

In 576 cases we had 282 male and 294 female patients. Our oldest patient was 54 years, the youngest a child of 3 years. Most of the male patients were between 21 and 25 years old, most of our female patients between 15 and 30 years old (Fig. 2). Relation of sex and side of operation is shown in Table II.

TABLE II. RELATION OF SEX TO SIDE OF OPERATION (576 CASES)

SEX	LEFT	RIGHT	BILATERAL	TOTAL	
				NO.	%
Male	130	144	8	282	49
Female	155	126	13	294	51
Total	285	270	21	576	100

STAGE OF DISEASE

Of 576 cases, twenty-one had bilateral pneumothorax and bilateral pneumonolysis. Of the remaining 555, a fair number of cases with bilateral disease had pneumothorax on the other side without thoracoscopy or pneumonolysis. Others with bilateral disease had interruption of the phrenic nerve, transthoracic suction drainage, or thoracoplasty on the opposite side. Table III shows the stage of disease in detail.

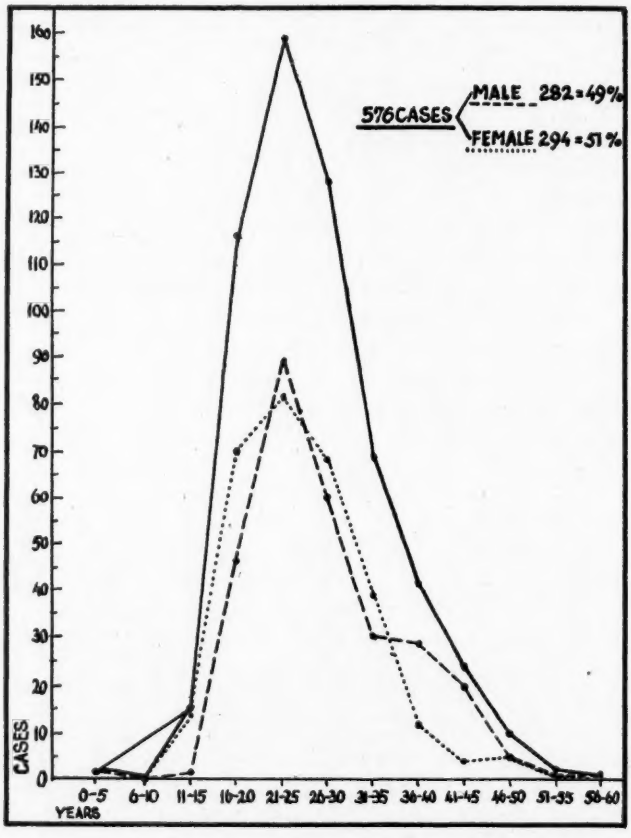

Fig. 2.—Distribution of patients according to sex and age.

Fig. 3 reveals that in the course of the years unilateral pneumonolysis was attempted in increasing number for moderately and far-advanced cases.

IMMEDIATE RESULTS

It is not easy to determine the result of a pneumonolysis, because complications may develop independently. The clinical result depends also on the

TABLE III. STAGE AND DISTRIBUTION OF DISEASE IN 576 PATIENTS WITH THORACOSCOPY AND PNEUMONOLYSIS

	MILD	MODERATELY ADVANCED	FAR ADVANCED	TOTAL
Unilateral thoracoscopy and pneumonolysis (555 cases)				
Unilateral disease	44	208	61	313
Bilateral disease	--	80	162	242
Bilateral thoracoscopy and pneumonolysis	--	4	17	21
Total	44 (7.6%)	292 (50.7%)	240 (41.7%)	576 (100%)

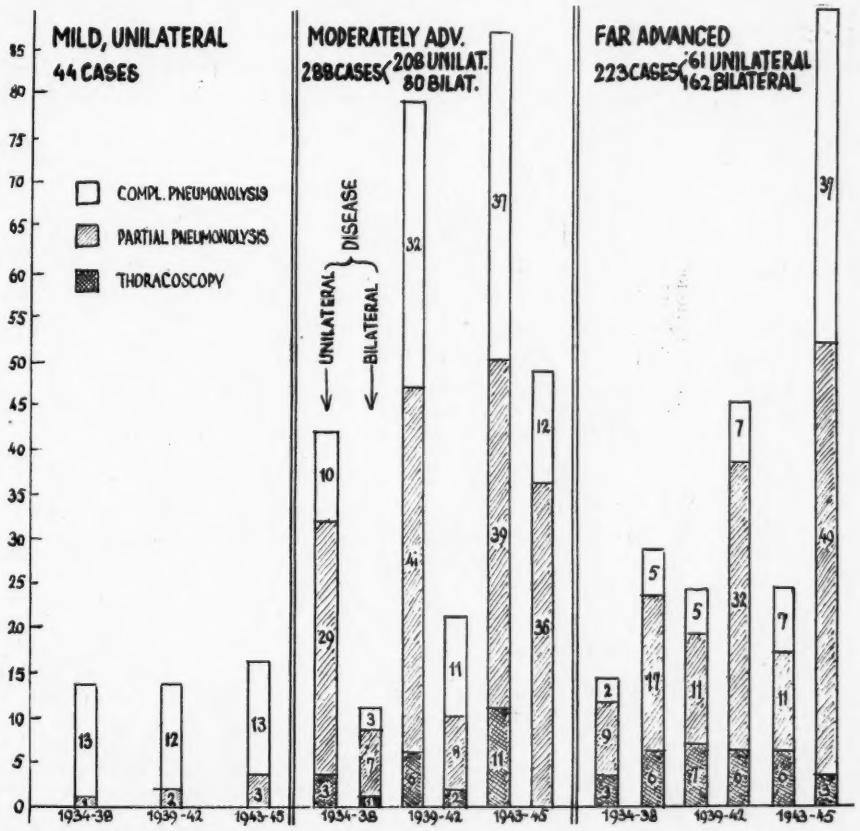

Fig. 3.—555 patients with unilateral thoracoscopy and pneumonolysis from 1934 to 1945.

condition of the other lung, so that only cases with the controlateral side free from disease or under control are able to give us a true picture of the result achieved. Pneumonolysis is not a therapeutic measure per se, but only an adjuvant to improving an inefficient pneumothorax. The simplest method of classification is to connect the immediate results with the effect of the pneu-

TABLE IV. IMMEDIATE RESULTS OF UNILATERAL THORACOSCOPY AND PNEUMONOLYSIS (479 CASES, 602 STAGES): RELATION TO NUMBER OF STAGES, TYPE OF OPERATION, FATE OF PNEUMOTHORAX A ND COMPLICATIONS WITHIN SIX MONTHS AFTER OPERATION

		OPERATION			FATE OF PNEUMOTHORAX FOR SIX MONTHS AFTER OPERATION										
						UNSATISFACTORY CASES									
							COMPLICATIONS								
STAGES	CASES	COMPLETE PNEUMONOLYSIS	PARTIAL PNEUMONOLYSIS	THORACOSCOPY	SATISFACTORY CASES	TOTAL	DANGEROUS NONCUTTABLE ADHESIONS	TENSION CAVITY	ADHESIVE PLEURITIS	FLUID	LEFT AGAINST ADVICE, REFUSED TREATMENT	SPONTANEOUS PNEUMOTHORAX BRONCHOPLEURAL FISTULA	EMPYEMA	DEATHS
I	385 (80.3%)	152	187	46	287 (74.5%)	98 (25.5%)	43	7	12	7	11	3	6	9	6
II	70 (14.6%)	19	47	4	50 (71.4%)	20 (28.6%)	7	1	3	2	1	0	1	4	2*
III	19 (4.0%)	3	16	0	13 (68.4%)	6 (31.6%)	4	0	1	0	0	0	0	1	0
IV	5 (1.1%)	3	2	0	4 (80.0%)	1 (20.0%)	0	0	1	0	0	0	0	0	0
Total	479 (100%)	177 (37.1%)	252 (52.5%)	50 (10.4%)	354 (73.8%)	125 (26.2%)	54	8	17	9	12	3	7	14	8

*One patient died after pneumonectomy performed four months after the second stage of partial pneumonolysis.

TABLE V. IMMEDIATE RESULTS OF BILATERAL THORACOSCOPY AND PNEUMONOLYSIS (21 CASES, 52 STAGES): RELATION TO NUMBER OF STAGES, TYPE OF OPERATION, FATE OF PNEUMOTHORAX AND COMPLICATIONS WITHIN SIX MONTHS AFTER OPERATION

		OPERATION			FATE OF PNEUMOTHORAX FOR SIX MONTHS AFTER OPERATION			
						UNSATISFACTORY (SIDES)		
STAGES	SIDES	COMPLETE PNEUMONOLYSIS	PARTIAL PNEUMONOLYSIS	THORACOSCOPY	SATISFACTORY (SIDES)	TOTAL	FLUID	ADHESIONS UNCUTTABLE
I	32 (76%)	25 (78.1%)	5 (15.6%)	2 (6.3%)	29 (90.6%)	3 (9.4%)	0	3
II	10 (24%)	4 (40%)	6 (60%)	0 –	9 (90%)	1 (10%)	1	0
Total	42 (100%)	29 (69%)	11 (26.2%)	2 (4.8%)	38 (90.5%)	4 (9.5%)	1	3

monolysis on the pneumothorax. A period of six months after operation seems to be sufficient to see the effect of the pneumothorax (closure of cavity, conversion of sputum, etc.). We may therefore stipulate the results in the following manner: thoracoscopy or pneumonolysis is satisfactory if a pneumothorax can be safely continued for a duration of six months; if a pneumothorax has to be abandoned within six months, the verdict should be unsatisfactory.

There were 500 patients with 521 sides in our series, observed for more than six months. 392 cases were satisfactory and 120 unsatisfactory, the ratio being 75.2 per cent and 24.8 per cent. Fig. 4 shows these figures in relation to the type of operation.

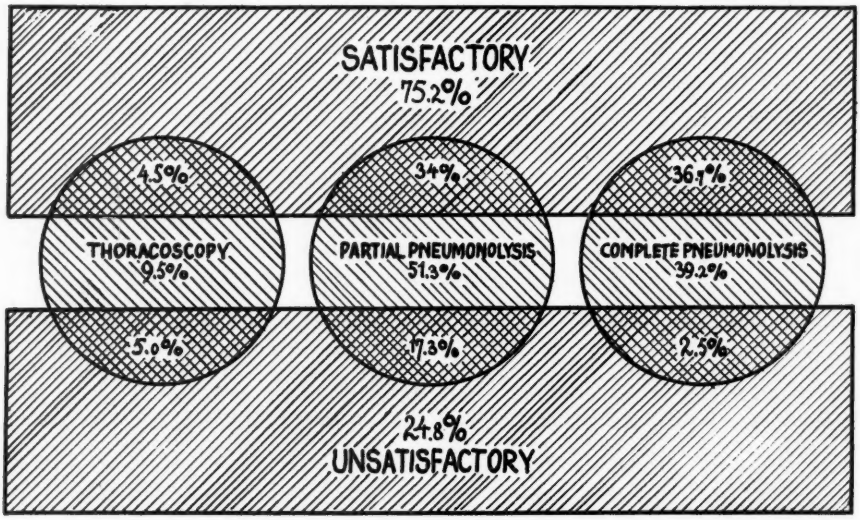

Fig. 4.—Results of complete or partial pneumonolysis and thoracoscopy in 500 patients (521 sides) six months after operation.

If we list the twenty-one cases with bilateral pneumonolysis separately, we arrive at the following figures: there were 90.5 per cent satisfactory and 9.5 per cent unsatisfactory results; the high percentage of success in these cases may be attributed to surgical daring in the face of far-advanced bilateral disease.

Of 479 cases of unilateral thoracoscopy or pneumonolysis, 385 had a one-stage operation, while the balance needed two or more stages. As Table IV illustrates, the satisfactory results diminish very little with the number of stages.

Of the twenty-one cases of bilateral operations, thirty-two sides had a one-stage operation and ten required two stages. There were no deaths nor major complications within six months in this group (Table V).

COMPLICATIONS

Complications may be due directly to the operation or may arise independently. It is very difficult to set a fast line. A spontaneous pneumothorax may develop two weeks after operation but the fistula may be in a place different from that of the severed adhesion (Fig. 5). We may classify all complications arising within four weeks after operation as postoperative, while for any complication after that interval the operation should not be blamed.

The main complications are the following: (1) considerable subcutaneous emphysema; (2) accumulation of fluid in the chest cavity; (3) hemorrhage; (4) spontaneous pneumothorax with or without persistent bronchopleural fistula; (5) empyema; and (6) adhesive pleuritis.

Subcutaneous Emphysema (See Table VI).—No treatment was given in the first and second degree. In the third degree, it was necessary to treat the

TABLE VI. INCIDENCE OF SUBCUTANEOUS EMPHYSEMA AFTER THORACOSCOPY AND PNEUMONOLYSIS

DEGREE	CASES	PER CENT
Slight	103	25.6
Moderate	34	8.5
Severe	7	1.8
None	256	64.1

underlying cause, which was usually increased intrathoracic pressure or tension pneumothorax (removal of air, administration of oxygen). Fig. 6 shows a case where the emphysema affected the whole chest wall, neck, and face. The eyelids could not be opened until the fifth day after operation.

Fluid Accumulation.—In 500 patients (521 sides), we encountered fluid in 291 instances. Tables VII and VIII show in detail the development and/or modification of fluid in relation to the postoperative period and the type of operation. In the multistage operations, only the fluid before the first stage and the condition after the last stage have been considered. There were 240 patients without fluid in the pleural space before operation, while four weeks to six months after the operation the number of patients without fluid increased to 374, or 73 per cent of all patients. Most of the patients with fluid for six months or longer after the operation had only partial pneumonolysis, or thoracoscopy showed adhesions unsuitable for cutting. Multiple stages more frequently caused fluid than single stages. If a multiple stage operation resulted in a complete pneumonolysis, the incidence of fluid almost reduced to that of single-stage operations.

Pleural effusions, especially larger ones and those following a hemorrhage, are usually accompanied by elevation of temperature. We noticed that there is a frequent relationship between the nature of the fluid and the height and duration of fever. Unless the fluid is hemorrhagic and due to operation, all other patients with high and long-standing fever will eventually develop em-

TABLE VII. DEVELOPMENT AND/OR MODIFICATION OF FLUID AFTER THORACOSCOPY AND PNEUMONOLYSIS: SINGLE STAGE OPERATION, 417 CASES

OPERATION	FLUID BEFORE OPERATION		FLUID WITHIN 4 WEEKS AFTER OPERATION					FROM 4 WEEKS TO 6 MONTHS AFTER OPERATION					
	AMOUNT	CASES	NONE	MINIMAL	MODERATE	CLOUDY	PUS	NONE	MINIMAL	MODERATE	LARGE	CLOUDY	PUS
Complete Pneumonolysis	None	99	69	24	6	0	0	91	5	2	0	1	0
	Minimal	74	12	49	12	1	0	49	18	2	2	0	3
	Moderate	4	0	3	1	0	0	4	0	0	0	0	0
	Total	177 (42.5%)	81 (45.7%)	76 (43%)	19 (10.7%)	1 (0.6%)	0 (0%)	144 (81.3%)	23 (13%)	4 (2.3%)	2 (1.1%)	1 (0.6%)	3 (1.7%)
Partial Pneumonolysis	None	78	61	9	8	0	0	71	5	0	0	1	1
	Minimal	92*	8	58	26	0	0	60	22	2	2	1	2
	Moderate	18	0	8	10	0	0	5	8	2	1	0	2
	Large	3	0	0	3	0	0	0	0	1	1	1	0
	Pus	1	0	0	0	0	1	0	0	0	0	0	1
	Total	192 (46%)	69 (35.9%)	75 (39.1%)	47 (24.5%)	0 (0%)	1 (0.5%)	136 (70.9%)	35 (18.2%)	5 (2.6%)	4 (2.1%)	3 (1.5%)	6 (3.1%)
Thoracoscopy	None	23	21	2	0	0	0	21	2	0	0	0	0
	Minimal	17	2	11	4	0	0	9	5	0	1	1	1
	Moderate	5	0	3	2	0	0	0	0	1	4	0	0
	Large	2	0	0	2	0	0	1	0	1	0	0	0
	Pus	1	0	0	0	0	1	0	0	0	0	0	1
	Total	48 (11.5%)	23 (47.9%)	16 (33.3%)	8 (16.7%)	0 (0%)	1 (2.1%)	31 (64.5%)	7 (14.6%)	2 (4.2%)	5 (10.4%)	1 (2.1%)	2 (4.2%)
Total		417 (100%)	173 (41.5%)	167 (40.1%)	74 (17.7%)	1 (0.2%)	2 (0.5%)	311 (74.7%)	65 (15.6%)	11 (2.6%)	11 2.6%	5 (1.2%)	11 (2.6%)

*Three patients died within four weeks after operation. (0.7 per cent.)

TABLE VIII. DEVELOPMENT AND/OR MODIFICATION OF FLUID AFTER THORACOSCOPY AND PNEUMONOLYSIS: MULTIPLE STAGE OPERATION, 104 CASES*

OPERATION	FLUID BEFORE OPERATION		FLUID WITHIN 4 WEEKS AFTER OPERATION				FROM 4 WEEKS TO 6 MONTHS AFTER OPERATION				
	AMOUNT	CASES	NONE	MINIMAL	MODERATE	LARGE	NONE	MINIMAL	MODERATE	CLOUDY	PUS
Complete Pneumonolysis	None	13	11	2	0	0	13	0	0	0	0
	Minimal	8	3	5	0	0	7	1	0	0	0
	Moderate	8	1	1	6	0	2	6	0	0	0
	Total	29 (27.9%)	15 (51.7%)	8 (27.6%)	6 (20.7%)	0 (0%)	22 (75.8%)	7 (24.2%)	0 (0%)	0 (0%)	0 (0%)
Partial Pneumonolysis	None	27	11	5	11	0	15	6	3	1	2
	Minimal	27	7	10	8	2	16	8	2	0	1
	Moderate	14	2	9	2	1	8	5	0	1	0
	Large	3	1	1	0	1	1	1	0	1	0
	Total	71 (68.2%)	21 (29.6%)	25 (35.2%)	21 (29.6%)	4 (5.6%)	40 (56.4%)	20 (28.2%)	5 (7%)	3 (4.2%)	3 (4.2%)
Thoracoscopy	Minimal	4	0	4	0	0	1	2	1	0	0
	Total	4 (3.9%)	0 (0%)	4 (100%)	0 (0%)	0 (0%)	1 (25%)	2 (50%)	1 (25%)	0 (0%)	0 (0%)
Total		104 (100%)	36 (34.6%)	37 (35.6%)	27 (25.9%)	4 (3.9%)	63 (60.5%)	29 (27.9%)	6 (5.8%)	3 (2.9%)	3 (2.9%)

*In order not to complicate the table, only the state of fluid before the first and after the last stage is considered.

pyema. If the onset of temperature is sudden, we have to suspect a traumatic lesion of the lung parenchyma (minute bronchopleural fistula), even if the fluid is clear for weeks and without demonstrable microorganisms. If such a pneumothorax is not abandoned at once, the change of the fluid into pus is the rule. Fluid with low-grade fever usually disappears if the underlying cause (adhesions, blood) is removed. Pleural effusion deriving from infection of the pleura does not show a sudden rise in temperature but causes a rather slow and steady elevation.

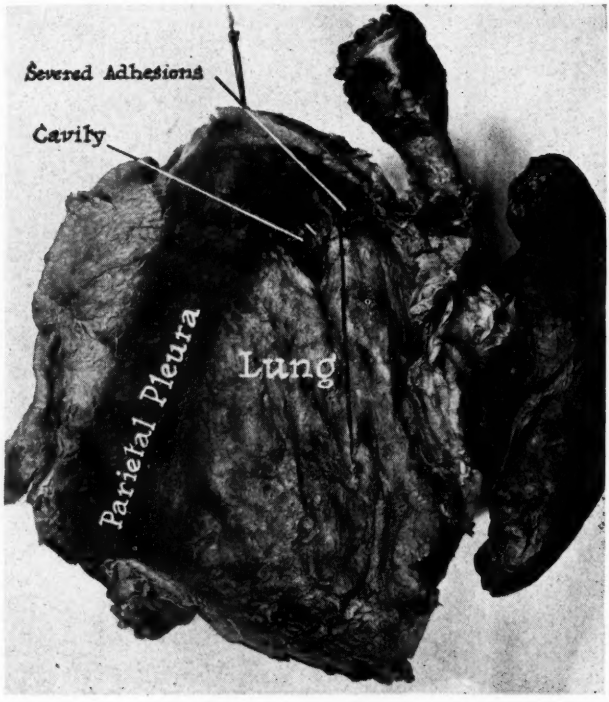

Fig. 5.—Rupture of cavity with spontaneous pneumothorax and fatal hemorrhage: cavity distant to severed adhesion.

Hemorrhage.—The incidence of hemorrhage in 754 operations was small and remained under the average reported by other authors. Table IX shows the number of hemorrhages.

There were no fatalities due to hemorrhage, and the loss of blood amounted to more than 100 c.c. in only eleven patients. In one patient the postoperative hemorrhage from a severed adhesion was so severe that the pleural cavity had to be packed. In another patient, a terminal hemorrhage occurred due to rupture of a cavity into the pleural space with no connection with a partial pneumonolysis two weeks before.

Prevention and treatment of hemorrhage during operation have already been discussed. Treatment of postoperative hemorrhage consisted in frequent and complete aspirations and partial replacement of fluid by air. Instillation of penicillin was carried out.

The incidence of hemorrhage falls as the experience of the operator increases.

Spontaneous Pneumothorax and Bronchopleural Fistula.—Out of fifteen cases of spontaneous pneumothorax, six were due to ruptured adhesions which contained lung tissue (in two instances the rupture occurred before operation).

Fig. 6.—Severe degree of subcutaneous emphysema after partial pneumonolysis.

Pneumonolysis was directly to be blamed in one case, leaving the residual adhesion under strain and with little support. Increasing of intrapleural pressure in order to "stretch" the adhesion aided greatly in the development of this complication.

Rupture of a tuberculous cavity was to be blamed in six instances. Three of them occurred in the four-week period after operation, two five weeks after, and one ten days before thoracoscopy. One patient had a complete pneumonolysis and in another at autopsy showed rupture of a cavity away from the partially severed adhesions. In both cases we probably dealt with tension cavities.

TABLE IX. INCIDENCE OF HEMORRHAGE IN THORACOSCOPY AND PNEUMONOLYSIS

HEMORRHAGE	CASES	SLIGHT	MODERATE	SEVERE
During operation				
Trocar puncture	3	3	0	0
Severed adhesions	10	6	4	0
Total	13	9	4	0
Postoperative	12	5	4	3*
Total	25	14	8	3
Per cent to all operations (754)	3.3	1.8	1.1	0.4

*One patient with a severe postoperative hemorrhage from a severed adhesion.

TABLE X. SPONTANEOUS PNEUMOTHORAX WITH OR WITHOUT BRONCHOPLEURAL FISTULA (15 CASES)

CASE	EXTENSION OF DISEASE		OPERATION	ONSET OF SPONTANEOUS PNEUMOTHORAX	PERSISTENT BRONCHOPLEURAL FISTULA	REASON	PX CONT. FOR 6 MONTHS	PN'X DISCONTINUED FOR		DIED WITHIN 6 MONTHS	REMARKS
								FLUID	EMPYEMA		
22	unl	fa	PPl	5 months ao	no	r res adh	yes	no	no	no	—
26	bil	ma	PPl	2 months ao	yes	r res adh	no	no	tuberculous	no	Pressures increased after PPl.
48	bil	fa	PPl	5 days ao (2nd st)	no	inj to l. by caut.	yes	no	no	no	Had two more stages: CPl.
71	bil	fa	PPl	6 weeks ao	yes	r res adh	no	no	mixed	no	—
74	bil	fa	PPl	3 days ao	yes	r cavity	no	no	tuberculous	no	Pressures increased after PPl.
78	bil	fa	CPl	6 days ao	yes	r cavity	no	no	mixed	no	Tension cavity; refused thoracoplasty
98	bil	fa	CPl	6 hours ao	no	inj to l. by caut.	yes	no	no	no	—
196	bil	fa	PPl	10 days bo	yes	r cavity	no	no	mixed	no	Bronchopleural fistula closed by endoscopic operation (Fig. 15)
230	bil	ma	PPl	3 months bo	no	r of adh	yes	no	no	no	Failure of having adhesion cut in time
247	bil	fa	PPl	2 weeks ao	yes	r res adh	no	no	no	yes	4 days after onset of spontaneous pneumothorax
324	unl	ma	PPl	10 hours ao	no	inj to l. by caut.	no	yes	no	no	—
358	unl	ma	Th	2 months bo	yes	r of adh.	no	no	tuberculous	no	Failure of having adhesion cut in time. Thoracoplasty
429	unl	ma	PPl	5 weeks ao	yes	r cavity	no	no	mixed	no	—
442	bil	fa	CPl	5 weeks ao	yes	r of tension cavity	no	no	tuberculous	no	—
447	bil	fa	PPl	2 weeks ao	yes	r of tension cavity	no	yes	no	yes	Died from hemorrhage into pleural space (Fig. 5)

Bil = bilateral; unl = unilateral; ma = moderately advanced; fa = far advanced; CPl = complete pneumonolysis; PPl = partial pneumonolysis; Th = thoracoscopy; ao = after operation; bo = before operation; r = rupture; res adh = residual adhesions; inj to l = injury to lung.

Direct injury to the lung during operation was the cause in the remaining three patients. The spontaneous pneumothorax occurred six hours, ten hours, and five days after operation.

Summing up, spontaneous pneumothorax occurred in seven instances as a postoperative complication in 521 operations. In four patients pneumothorax could be continued, eight were followed by empyema (four mixed infections), one pneumothorax was discontinued because of effusion, and two patients died within six months after operation. Table X gives a short review of these cases.

Treatment of spontaneous pneumothorax relieved the intrapleural pressure by aspiration of air or continuous underwater drainage. In later years we have used instillations of penicillin.

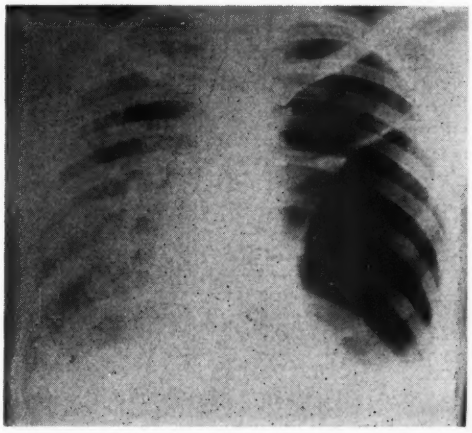

Fig. 7.—Spontaneous pneumothorax six days old, treated by closure of a persistent bronchopleural fistula.

In one of our patients we were able to close the bronchopleural fistula by endoscopic procedures. The case history is as follows:

The patient, a 30-year-old female with bilateral, far-advanced disease, was admitted to the hospital six days after the onset of a tension pneumothorax with a temperature of 104° F. Adhesions were found in the x-ray. They were considered very dangerous as removal of air at several occasions did not change the positive pressure. A thoracoscopy four days after admission visualized a shelf adhesion which was not completely cuttable. (Fig. 7.) A small bronchopleural fistula was found near to the insertion of the adhesion. It was possible to fashion a pedicle flap from the adhesion and to plug it into the small hole. Partial coagulation of the graft baked the latter to the visceral pleura with the effect of hermetic closure of the fistula. Tension pneumothorax subsided and the pressure remained negative. Contamination of the pleural cavity with *Staphlococcus aureus* and acid-fast organisms had already taken place. Onset of an empyema could not be prevented. Frequent aspiration, irrigation, and instillation of penicillin helped to dry up the empyema completely. One year afterward, the lung was completely re-expanded. No thoracoplasty was contemplated on account of the bilateral disease and a low vital capacity. Only a temporary interruption of

the phrenic nerve was carried out, in order to promote the re-expansion of the lung. This patient, although having Gaffky 6 (Gaffky 9 at admission), is still alive (4 years since incident) and put on 30 lb. of weight. Later we saw an x-ray taken two months before the onset of the spontaneous pneumothorax. It revealed two interlocking giant cavities corresponding to the place where the fistula was found. X-rays and tomograms after reexpansion did not show these cavities.

Empyema.—In order to be practical, any collection of pus in the pleural cavity should be considered an empyema, whether or not organisms can be found. With a few exceptions, the nature of the empyema can be ascertained from the nature of the pulmonary lesion.

In our series of 500 cases (521 sides, 654 operations) there were seventeen instances (3.3 per cent) where an empyema developed within six months after operation without pre-existing purulent fluid. There was no case of empyema in the 21 cases with bilateral operations. Table XI shows the relationship between onset of empyema and type of operation.

TABLE XI. TYPE OF OPERATION AND ONSET OF EMPYEMA

	ONSET WITHIN 4 WEEKS			4 WEEKS TO 3 MONTHS		3 TO 6 MONTHS		
TYPE OF OPERATION	SPONTANEOUS PNEUMOTHORAX	INJURY TO LUNG	SPREAD	SPONTANEOUS PNEUMOTHORAX	SPREAD	SPONTANEOUS PNEUMOTHORAX	SPREAD	TOTAL
Complete pneumonolysis	1	0	0	0	0	1	2	4
Partial pneumonolysis	1	1	1	3	2	0	3	11
Thoracoscopy	0	0	1	0	0	0	1	2
Total	2	1	2	3	2	1	6	17

In one of these patients (No. 69) the empyema could be traced directly to a lung injury. In the two patients with spontaneous pneumothorax within the first four weeks, it was due to the rupture of a cavity. In one patient (spread of disease within four weeks) pneumothorax should never have been attempted (tuberculous bronchopneumonia with repeated severe hemoptyses).

There were four additional cases of empyema where the onset could be traced back to pre-existing lesions of the pleural cavity and where the operation did not cause it.

Acid-fast bacilli were found in sixteen cases. Five cases were mixed infections (four of them due to persistent bronchopleural fistula).

Three patients died within six months but none during the first four weeks. Table XII gives a review of all twenty-one empyema cases encountered in our series.

The accepted treatment of tuberculous empyema is a complete thoracoplasty and, in rare instances, a decortication of the lung. Thoracoplasty was performed in all those cases where the other side was free from disease or at least under control (ten cases). In mixed infections, penicillin was used since 1943 with gratifying results: 50,000 units were instilled every other day after

complete removal of the pus by special posturing (Kunstler). Thoracotomy and open or closed drainage had to be done in two instances.

Adhesive Pleuritis.—Adhesive pleuritis may bring the most promising artificial pneumothorax to a sudden end. Maxwell, in a detailed study of readhesions, divides the postoperative obliteration of the pleural space into three groups: (1) acute obliteration within forty-eight hours, often total; (2) secondary obliteration which takes place within a week or two. It is more localized with predilection of the posterior mediastinum and the scapular zone. Faulty operative posture and inadequate control of pressures are claimed to be important causal factors; (3) late obliteration. It is due to pleural effusion and re-expansion of the lung with disappearance of the fluid.

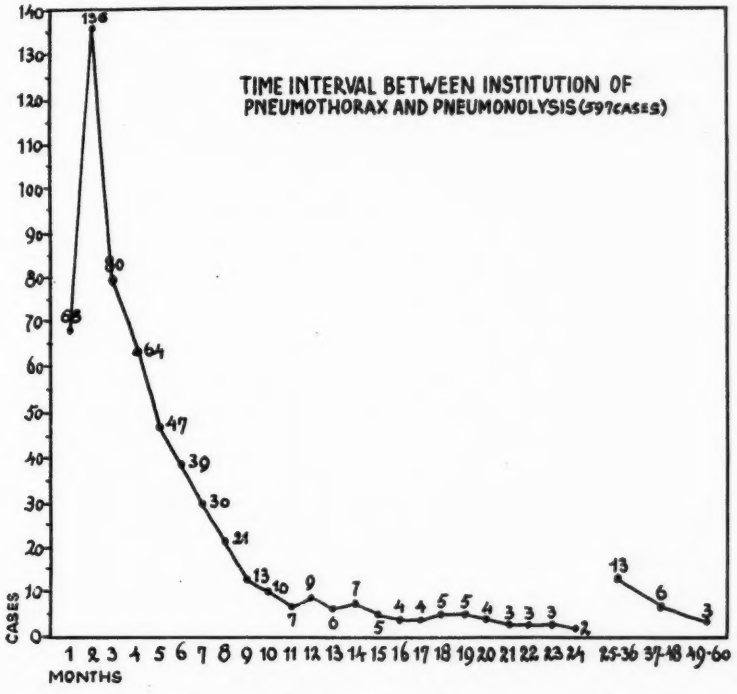

Fig. 8.—Time interval between institution of pneumothorax and pneumolysis (597 cases).

Maxwell found the incidence of readhesions in the literature 1.3 per cent while Moore calculated in base of his review only 0.29 per cent. In our cases we had seventeen cases of readhesions within the first month (0.9 per cent) and the pneumothorax had to be abandoned. In one case we tried to cut the adhesions four times, but according to the operation record the adhesions grew faster than the surgeon could cut them. In some of the seventeen cases we can attribute the failure to our negligence or the patient's, in that the refills

TABLE XII. EMPYEMATA WITHIN SIX MONTHS AFTER PNEUMONOLYSIS OR THORACOSCOPY (21 CASES)

CASE	EXTENT OF DISEASE	OPERATION	ONSET	CAUSE	BACT. OF FL. ACID F.	BACT. OF FL. MIXED	SPUT. CONV.	LUNG REEXPANDED	DIED (6MO.)	REMARKS
10	bil fa	PPl 2st	4 weeks ao	spread pleura	+	–	bil	no	yes	Tb bronchopneumonia, severe hemoptysis, not controlled by pneumothorax
16	bil fa	PPl	4 months ao	spr. to pleura	–	–	bil	no	no	
21	unl fa	Th	6 months ao	spr. to pleura	–	–	3 months ao	no	no	Phrenic nerve interruption
26	bil ma	PPl 2st	2 months ao	r. res. adh.	–	–	4 months ao	no	no	
29	bil fa	Th 3 rd Stage	2 weeks ao	Infection of pleura bo	+	–	2 weeks after 1st st	?	no	Moderate fluid bo. 3rd stage only Th.: lung covered with fibrin after first stage left against advice after last stage.
59	bil fa	PPl	2 months ao	?	+	–	bil	?	no	No sufficient record
63	bil fa	Th	2 months ao	spr. t. pleura	+	–	6 months ao	no	no	Had exudative pleuritis before operation. Thoracoplasty
64	bil fa	CPl	6 months ao	spr. t. pleura	+	–	neg. bo	yes	no	
66	unl ma	Th	3 weeks ao	spr. t. pleura	–	–	neg. bo	no	no	Had thickened pleura before operation, minimal fluid
69	unl fa	PPl	2 weeks ao	inj. t. lung	+	–	no	no	yes	Cavity collapsed, refused thoracoplasty. Fluid before operation
71	bil fa	PPl	8 weeks ao	sp. px. 6 w. ao	+	+	bil	no	no	Rupture residual adhesions. Thoracotomy, Thoracoplasty.
74	bil fa	PPl	3 weeks ao	sp. px, br-plf.	+	–	bil	no	no	Fluid before operation
78	bil fa	CPl	2 months ao	sp. px. 6 d. ao	+	+	bil	no	no	Rupture of cavity. Refused thoracoplasty.
94	unl fa	CPl	3 months ao	spr. t. pleura	+	–	5 months ao	no	no	
104	unl fa	PPl 2st	2 months ao	spr. t. pleura	–	–	1 week a. 2nd st.	no	no	Minimal fluid before operation. Thoracoplasty at 6 months.
108	bil fa	PPl 2st	3 months ao	spr. t. pleura	+	+	neg. after 2nd st.	yes	no	Penicillin local, thoracoplasty.
196	bil fa	PPl	10 days bo	sp. px, br-pl. fist. r. cav.	+	+	bil	yes	no	Closure of fistula by endoscopic operation, phrenic nerve interruption, penicillin into pleural cavity.
217	bil fa	PPl	3 months ao	spr. t. pleura	+	–	bil	yes	yes	Exudative pleuritis, 102 temperature bo. Intest. tb., poor risk.
358	unl ma	Th	before Th.	r. adhesions	+	–	6 months ao	no	no	Lesion seen at thoracoscopy. Thoracoplasty. Pneumothorax 10 ms. duration bo
429	unl ma	PPl	5 weeks ao	sp. px. r. cav.	+	+	neg. bo	no	no	
442	bil fa	CPl	3 months ao	sp. px. r. cav.	+	–	yes	no	no	Pneumothorax discontinued before rupture of tension cavity

bil = bilateral; unl = unilateral; ma = moderately advanced; fa = far advanced; CPl = complete pneumonolysis; PPl = partial pneumonolysis; Th = thoracoscopy; ao = after operation; bo = before operation; r = rupture; res. adh. = residual adhesions; inj. to l. = injury to lung; sp. px. = spontaneous pneumothorax; br-pl. fist. = bronchopleural fistula.

were not given at the right interval or in right amounts. Wrong posturing may favour readhesions, but, as after thoracoplasties, we paid more attention to the controlateral "good" side and ordered posturing accordingly. In the majority of our cases, however, the re-expansion was due to the pathological process and could not be avoided. Most of these patients had subsequent thoracoplasty.

MORTALITY

Of the 500 consecutive patients considered in the postoperative phase, three died within four weeks after operation and five died within six months. They all had partial pneumonolysis performed and are considered in detail in Table XIII.

TABLE XIII. FATAL CASES WITHIN SIX MONTHS AFTER THORACOSCOPY AND PNEUMONOLYSIS

CASE	EXTENT OF DISEASE	OPERATION	TIME OF DEATH	IMMEDIATE AND REMOTE CAUSE OF DEATH
10	bil fa bronchopneumonia, cavities*	PPl 2 stages	2 months after operation 2nd stage	General downhill course, Gaffky VIII; spread to pleura; empyema
25	bil fa cavity r. base, phrenic paralysis	PPl 2 stages	4 months after operation 2nd stage	Pneumonectomy in 1941, died postoperatively.
69	unl fa cavity fluid	PPl	3 months after operation	Injury to lung during operation, empyema, refused thoracoplasty
138	bil fa cavities; 9 rib thoracoplasty on opposite side; Positive sputum, hemoptysis from pneumothorax side	PPl	2 weeks after operation	Pneumonitis, atelectasis, anoxemia; bronchoscopic aspirations without effect.
217	bil fa, intestinal tb; cavities	PPl	3 months after operation	Spread to pleura, empyema; general downhill course.
247	bil fa cavities	PPl	18 days after operation	Rupture of residual adhesions, spontaneous pneumothorax, bronchopleural fistula.
310	bil fa fluid, cavities	PPl 2 stages	2 months after operation	General downhill course, Gaffky VIII.
447	bil fa cavities	PPl	2 weeks after operation	Rupture of tension cavity, terminal hemorrhage from cavity into pleural space. Spontaneous pneumothorax and bronchopleural fistula (Fig. 5).

*See footnotes on abbreviations on Tables X and XII.

Only four cases can be directly attributed to the operation, which gives a six-month case mortality of 0.8 per cent and a stage mortality of 0.6 per cent.

One of these four patients with far-advanced bilateral disease had a nine-rib thoracoplasty for cavitary lesion on one side. The operation resulted in a complete closure of the cavity which was confirmed by tomogram. A pneumothorax was instituted on the other side in order to control the positive sputum and severe hemoptyses. Adhesions did not allow a selective collapse and partial pneumonolysis was performed. The patient died from anoxemia due to pneumonitis and partial atelectasis unrelieved by repeated bronchoscopic aspirations, two weeks after operation.

The second patient died from empyema three months after operation. The empyema was due to direct injury to the lung during partial pneumonolysis.

The third patient died after eighteen days of anoxemia due to uncontrollable spontaneous pneumothorax caused by rupture of residual adhesion.

The last one had a terminal hemorrhage from a blow out of a cavity two weeks after partial pneumonolysis (Fig. 5).

In the other four patients the pneumonolysis had no connection with the death. Three of them died from further spread of disease and one after pneumonectomy (1941) four months after an unsuccessful pneumonolysis.

Table XIV gives the percentage of case and stage mortality.

TABLE XIV. CASE AND STAGE MORTALITY WITHIN SIX MONTHS

	STAGE MORTALITY (%)	CASE MORTALITY (%)
All cases	1.2	1.6
Deaths due to operation	0.6	0.8

COMMENT

G. R. Howell considered a consecutive number of pneumothorax cases of the Royal Edward Laurentian Hospital and came to the conclusion that out of every 100 attempted pneumothoraces only sixteen showed a perfect result without evident adhesion. In 19 per cent no free pleural space was found and in 13 per cent the lung was adherent to the chest wall over such a broad area that the pneumothorax was immediately abandoned. In 52 per cent of the cases adhesions were found, but the pneumothorax was continued for various lengths of time. It is this group of patients that came to thoracoscopy and pneumonolysis.

While in earlier years not all of Howell's 52 per cent group came up for thoracoscopy, we later followed the policy of attempting pneumonolysis in every case in this group. We cannot exclude the presence or absence of adhesions by x-ray alone. We therefore operated on patients in the absence of radiologically demonstrable adhesions when (1) fluid covered the costophrenic angle (56 per cent of all cases had fluid from minimal to large amounts previous to operation), (2) cavities did not collapse, and sputum remained positive in unilateral cases, (3) repeated hemoptyses with the other side free from disease or under control, (4) spontaneous pneumothorax with uncontrollable bronchopleural fistula.

If a cavity remains open in the absence of adhesions or after complete pneumonolysis, it should be suspected of being a tension cavity. In such cases we abandoned the pneumothorax immediately by withdrawing air or by oxygen lavage.*

Pneumothoraces which develop fluid and high fever should also be immediately abandoned, if the temperature does not subside within a week after operation. They are potential candidates for an empyema.

75.2 per cent of our patients had a satisfactory result. Complete and partial pneumonolysis was performed on them at approximately the same rate (36.7 per cent and 34 per cent). Of the 24.8 per cent unsatisfactory cases,

*See illustrations in Surg., Gynec. & Obst. 78: 254, 1944.

however, partial pneumonolysis forms the major part, with 17.3 per cent. The problem starts with those patients on whom a complete pneumonolysis cannot be done in one stage. In cases with mild, moderately, or far-advanced unilateral disease, we prefer to abandon the pneumothorax (and do thoracoplasty, lobectomy, pneumonectomy), if a complete pneumonolysis or a safe partial pneumonolysis cannot be done in one stage.

There remains the group of cases with far-advanced bilateral disease. In spite of greater risks, pneumonolysis may be the only chance to achieve a satisfactory collapse. The number of patients with far-advanced bilateral conditions coming up for pneumonolysis increased rapidly in the ten-year period, while the mild and moderately advanced cases showed an increase to a much lesser extent.

Table XV illustrates the higher incidence of complications in the far-advanced and bilateral groups.

TABLE XV. MAJOR COMPLICATIONS IN RELATION TO STAGE AND DISEASE

COMPLICATION*	UNILATERAL (%)	BILATERAL (%)	MILD AND MODERATELY ADVANCED (%)	FAR ADVANCED (%)
Spontaneous pneumothorax	28.5	71.5	35.7	64.3
Empyema	33.3	66.7	19.1	80.9

*Within six months after operation.

It is somewhat difficult to appreciate the risk connected with the multistage operations. Tables IV and V show that the satisfactory results in the multistage operations are almost equal in proportion to those with a single stage operation. The surgical interference is apparently of minor importance and the great risk is rather represented by the waiting period from one stage to the other. Quite a number of residual adhesions ruptured in the interval.

We may therefore say that multistage operations should be considered only, if residual adhesions are considered safe, if the patient remains under close supervision during the interval, and if intrapleural pressures are well adjusted and "stretching" strictly avoided.

We may here also discuss the question of the time interval between institution of artificial pneumothorax and the operation. This was the subject of the presidential address given by J. E. H. Roberts at the meeting of the British Tuberculosis Association in 1944. He pointed out that thoracoscopy should be performed as soon as adhesions are diagnosed, or, in other words, no definite time period should be stated.

We agree with Mr. Roberts. We try to operate as soon as direct or indirect diagnosis of adhesions is made. Fig. 8 illustrates that most of our adhesions were divided within the second month after institution of pneumothorax. The shortest time between initial pneumothorax and thoracoscopy was ten days, the longest, five years.

There is a widespread opinion that within the first weeks the available airspace is too small for the introduction of the instruments. It is true that even oblique x-rays may show only a small marginal zone of air although the

patient is taking 300 to 500 c.c. twice weekly without considerable change of the intrapleural pressures. In these cases the lung is always collapsed in an anterior-posterior direction due to lateral or posterior adhesions with a large air space in front of the lung. Expiratory stereoscopic x-rays are very useful. Very often a shift of the anterior mediastinum is evident and significant. At rare occasion the airspace may be situated posteriorly with consequent shift of the posterior mediastinum.

CONCLUSIONS

From discussing the results in our series of 500 cases with 654 operations, the following conclusions may be drawn:

1. Every patient with pneumothorax should be closely guarded against possible complications and submitted to thoracoscopy as soon as direct or indirect signs of adhesions are detected. Indirect signs are (a) presence of fluid in costophrenic angle, (b) insufficient collapse of diseased lung tissue, cavities, and persistence of positive sputum (unilateral cases or controlateral side under control) in spite of regular and apparently sufficient refills, (c) partial shift of the mediastinum. Direct signs of adhesions may be recognized only on a later date, but expiratory x-rays, oblique stereo x-rays, tomograms, and outlining of oesophagus by radioopaque substances make it possible to establish such a diagnosis before direct signs such as indicated are available.

2. Thoracoscopy is a harmless procedure; the only potential complications which need be considered are subcutaneous emphysema and hemorrhage from the trocar puncture. Both may be reduced by technical modifications such as those outlined. General anaesthesia is of definite disadvantage and local infiltration with novocaine is the choice.

3. Adhesions should be divided if (a) they are responsible for an insufficient collapse of the diseased part of the lung, (b) they are suitable for separation without anticipation of complications as injury to lung tissue, rupture of residual adhesions and perforation of cavities or hemorrhage.

4. Adhesions of major caliber or multiple small adhesions should not be divided if they make possible a selective collapse, especially in bilateral cases.

5. Multistage operations should be attempted only if (a) no other procedure is feasible, (b) intrapleural pressures are well controlled and (c) prevailing circumstances permit a further stage at any time considered optimal for the individual case.

6. The determination of vital capacity or of the oxygen consumption of each lung separately (bronchospirometry) should be done in the presence of bilateral disease. Bronchoscopy should be performed in all cases before institution of pneumothorax, if this is possible.

7. Pneumothorax should be abandoned immediately if (a) important adhesions are not suitable for cutting and other procedures (thoracoplasty, lobectomy, pneumonectomy) possible, (b) fluid and high fever develop and persist for more than a few days, (c) conditions which predispose to the development of empyema are present, especially injury to the lung by the cautery, rupture of residual adhesions, or of a cavity and extensive active disease of the lung,

(d) oxygen consumption is below the minimum compatible with light exercise, (f) tension cavities are present.

8. The only exceptions to paragraph (7) are those bilateral far-advanced cases with severe repeated hemoptyses originating most likely from the pneumothorax side.

9. The instruments best serving the purpose are in our opinion those designed by Coryllos. The diathermic cautery may be superior to the live cautery in some aspects but is by no means the key to a successful operation.

10. How often does pneumonolysis convert a pneumothorax which is questionable or dangerous due to adhesions, into a safe satisfactory collapse of the lung? In our series with 500 cases and 654 stages conversion occurred in 75 per cent. This result raises the number of safe pneumothoraces from sixteen to fifty-five out of every 100 cases attempted, i.e., an increase of more than three times.

11. The principal errors committed in our series were (a) unplanned timing of first and subsequent Jacobaeus operations, (b) failure to abandon pneumothorax in certain cases as outlined above, (c) attempted pneumonolysis in cases with low vital capacity, active disease of the pleura, tuberculous bronchopneumonia, tension cavities, (d) inadequate control of pneumothorax after operation, (e) injury to lung during operation, (f) extraneous factors not dependent on medical and surgical competency.

(12) Favourable results from pneumonolysis are, *ceteris paribus*, dependent on the experience and the skill of the operating surgeon. Teaching of the technique is rather difficult, due to the endoscopic nature of the procedure. A binocular telescope may be helpful for demonstration of the technique to the pupil.

We have prepared a motion picture (16 mm. color), taken first on cadavers, later on the living patient, and believe that this is a partial answer to the problem of teaching.*

SUMMARY

The technical results of thoracoscopy and pneumonolysis in 500 cases with 654 stages are discussed, the advantages and risks of the operation described in detail, and suggestions made in order to prevent the relatively frequent complications. No attempt has been made in this paper to follow up these cases for more than six months, and no conclusions were drawn as to the ultimate clinical results.

REFERENCES

Alexander, John: The Collapse Therapy of Pulmonary Tuberculosis, Springfield, Ill., 1937, Charles C. Thomas.

Coryllos, Pol. N.: J. THORACIC SURG. 7: 48, 1937.

Coryllos, Pol. N., and Ornstein, G. G.: Management of Bilateral Cavernous Pulmonary Tuberculosis, J. THORACIC SURG. 5: 337, 1936.

Howell, R. G.: Immediate Results of 200 Consecutive Cases of Attempted Artificial Pneumothorax, Ann. Meet. Cdn. Med. Ass., Que. Div., Montreal, January, 1945.

Howell, R. G.: Personal Communication.

Jacobaeus, H. C.: Die Verwendung der Thorakoskopie und Laparoskopie für die Diagnose tuberkulöser Erkrankungen, Handbuch der Tuberkulose 1: 731, 1914.

*Limited space does not allow us to go into details of the photographic technique and illustrations. They will, however, be the subject of a separate article, to be published later.

Jacobaeus, H. C.: The Cauterization of Adhesions in Artificial Pneumothorax Treatment of Pulmonary Tuberculosis. Am. Rev. Tuberc. **6**: 871, 1922.

Kunstler, W. E.: Immediate Results of 200 Cases of Thoracoscopy and Pneumonolysis, Ann. Meet. Cdn. Med. Ass., Quebec. Div., Montreal, January, 1945.

Kunstler, W. E.: A Source of Danger in Thoracentesis and Its Prevention, Canad. M. A. J. **53**: 552, 1945.

Matson, R. C.: Intrapleural Pneumonolysis Severing Adhesions in Artificial Pneumothorax, Goldenberg, Clinical Tuberculosis ed. 3, Philadelphia, 1942, F. A. Davis Co., Vol. 1, p. D 71.

Maxwell, R. J. C.: Readhesions After Intrapleural Cauterization, J. THORACIC SURG. **14**: 194, 1945.

Moore, J. A.: Intrapleural Pneumonolysis, J. THORACIC SURG. **3**: 276, 1934.

Roberts, J. E. H.: Presidential Address, Brit. Tubere. Ass. 1944.

Unverricht, W.: Die Thorakoskopie als Hilfsmittel für die endopleurale galvanokaustische Durchtrennung von Pleurasträngen, sowie für eine neue Lokalisationsmethode extrapleural anzugreifender Adhäsionen, Zeitschr. f. Tuberk, **36**: 267, 1922.

Vineberg, A. M., and Kunstler, W. E.: The Role of Intracavity Pressures in Collapse Therapy for Pulmonary Tuberculosis, Canad. M. A. J. **46**: 329, 1942.

Vineberg, A. M. and Kunstler, W. E.: The Determination and Treatment of Pressure Cavities in Pulmonary Tuberculosis, Surg. Gynec. & Obst. **78**: 245, 1944.

AN UNUSUAL HAMARTOMA (SO-CALLED CHONDROMA OF THE LUNG)

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ALTHOUGH small, so-called, chondromas in the lung are occasionally encountered as incidental findings at autopsy or as accidental findings in routine x-rays of the lungs; the occurrence of relatively large, intrapulmonary, cartilaginous tumors which produce clinical symptoms is distinctly uncommon. Ordinarily, so-called chondromas vary from a few millimeters to 3 to 4 cm. in diameter, but a few tumors varying from 5 to 15 cm. in diameter have been encountered. They may be subpleural in position, may lie near the hila or elsewhere in the lung, and are firm, dense, inclined to be somewhat lobulated, sharply demarcated, and gray and glistening upon section. The smaller tumors rarely, if ever, produce clinical symptoms.

These so-called chondromas are not pure cartilaginous tumors but contain abnormal mixtures of elements normally encountered in bronchial wall. They are, therefore, more properly referred to as hamartomas[1] or hamartoma chondromatosum pulmonis.[2]

Although hamartoma of the lung is usually considered as a benign, slow-growing tumor, it is possible that malignant changes may occur. The hamartoma in this instance is unusual because of the size of the tumor, its histologic character, and the fact that it produced symptoms and a roentgenologic appearance which strongly simulated the usual bronchiogenic carcinoma.

CASE REPORT

H. E., a salesman, aged 49 years, developed an insidious cough during February, 1943. This complaint was soon accompanied by mucopurulent expectoration and moderately severe, more or less constant, pain in the left chest posteriorly. Three months later, when he first came under the care of Dr. D. H. Ballon and one of us (H. C. B.), roentgen-ray studies revealed a large, circumscribed, slightly lobulated, dense mass in the middle and posterior portions of the left chest (Fig. 1). The left diaphragm moved but little with respiration. He was twice given bronchoscopy by Dr. D. H. Ballon at the Royal Victoria Hospital. On May 19, 1943, tissue was removed from what was thought to be a papillomatous mass in the left main bronchus. Dr. T. R. Waugh reported that the tissue consisted, for the most part, of mucous gland structures and blood clot within fibrous and muscular tissue partly surmounted by a rather fragmented, pseudostratified, columnar epithelial layer. In a few places this gave way to stratified squamous cells. Small nests of mature cells of epithelial type were present just beneath the lining cells. Dr. Waugh was of the opinion that the findings did not permit of a diagnosis of malignancy. Bleeding and profuse purulent discharge precluded the taking of a biopsy during the course of the second bronchoscopic examination carried out ten days later. Previously febrile (90 to 100° F.) on June 1, 1943, he developed a tem-

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perature of 104° F. and a fluid level was noted in the left pleural cavity. The left diaphragm could no longer be visualized. There was increased hilum detail on the right. He was referred for admission to the Jewish General Hospital. The clinical diagnosis throughout was suppurating carcinoma of the left lung.

Physical Examination.—The patient, of sallow complexion, had obviously lost considerable weight and was perspiring profusely. There was decreased respiratory excursion of the left chest. The percussion note was impaired over the greater part of the left posterior chest. Over its midportion, breath sounds were diminished. There were numerous moist râles at the base of the left lung, and scattered râles at the right base. The blood pressure was 164/80.

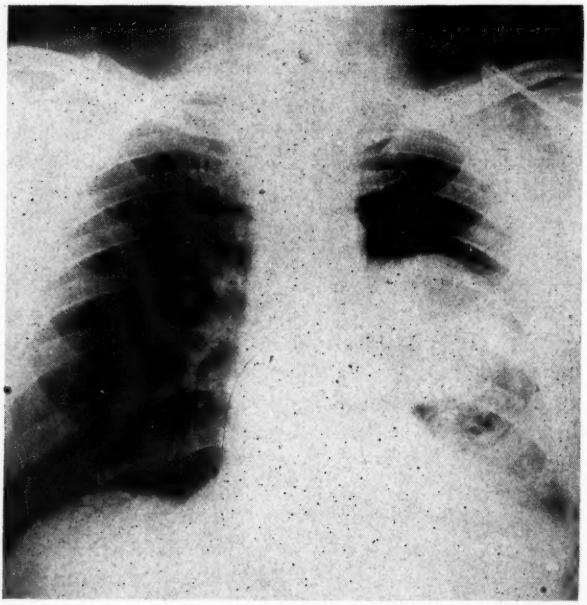

Fig. 1.—Dense, lobulated mass occupying the midportion of the left lung. The heart and mediastinal contents are drawn over slightly to the left. There is a small pleural effusion at the left base.

Laboratory Findings.—The urine showed a trace of albumin and an occasional pus cell. A hemogram showed a slight anemia; the leucocytes numbered 16,000 per cu.mm. The blood Kline reaction was negative. Repeated examinations of the sputum were negative for acid-fast bacilli. Cultures of the sputum revealed the presence of *Streptococcus hemolyticus* and *Staphylococcus aureus hemolyticus.*

Clinical Course.—A thoracentesis was performed on June 4, 1943, when a few cubic centimeters of blood-tinged fluid were aspirated. This fluid was sterile. A second aspiration on June 8 yielded a small quantity of slightly turbid, amber-colored fluid. Smears of the centrifugalized pleural fluid revealed moderate numbers of agglomerated cells, the majority of which could be recognized as polymorphonuclear leucocytes. The masses of cells showed marked degenerative changes to an extent which prevented recognition or study of their finer details. No cells could be identified as tumor cells.

Large doses of sulfathiazole did not appreciably influence either the toxic clinical manifestations or the underlying lesion. These, together with the roentgenologic evidence on

June 7, 1943, of a spill over to the contralateral lung, precluded an exploratory thoracotomy and pneumonectomy which had been contemplated. During July the patient developed hemoptysis daily. His voice became hoarse. His hemoglobin was now 56 per cent. He was subjected to a course of deep x-ray therapy by Dr. D. Berger and received 4662r without benefit.

The third and last admission was on Sept. 23, 1943, when the patient was semistuporous, unable to swallow, and had a right-sided facial weakness. His temperature was 102° F., pulse 152, and respirations were 40 to the minute. The abdominal reflexes were doubtful; there was a questionable Babinski on the right. The leucocytes numbered 14,300 per cubic millimeter. X-ray examination of the chest one day previous to his death revealed almost uniform obscuration of the left hemithorax. The cardiac shadow was observed to be shifted to the left. A left-sided, apical, paramediastinal mass was also noted. He expired on Sept. 25, 1943, eight months after the onset of his complaints.

For at least five months of this period he had exhibited signs of severe bilateral lung suppuration. His sputum, at first mucopurulent, was subsequently blood-tinged and only latterly slightly foul. The final clinical diagnosis was carcinoma of the left lung with chronic pneumonitis (abscess formation and bronchiectasis), pleural effusion, and metastases to the brain.

Necropsy Findings.—Necropsy was performed one hour and ten minutes after death.

No external abnormalities were noted. The right thoracic cavity contained a voluminous crepitant lung of a mottled gray, red, and black color. No fluid or adhesions were present on this side. Posterolaterally, the left lung was bound by dense, fibrous adhesions. The left base was adherent to the diaphragm. In the left lung a large, irregularly lobulated and globular mass of bony consistency could be palpated. This mass was continuous with a left paramediastinal mass which pulled the trachea slightly to the left. The pericardial sac contained 200 c.c. of clear fluid having a specific gravity of 1.005.

The two lobes of the left lung were fused and no interlobar division could be identified either externally or on section. The left main bronchus was slightly widened and at its division into upper and lower lobe bronchi, it was noted that the left upper lobe bronchus was pushed medially and anteriorly by the large, bony-hard mass which lay in what was probably the left lower lobe. The lumen of the left upper lobe bronchus appeared slightly narrowed and was filled with thick, gray, odorless pus. The mucous membrane of this bronchus was gray and intact. No ulceration or erosion was noted. The left upper lobe bronchus rode over the underlying bony mass.

The lumen of the left lower lobe bronchus was narrowed to a slit by the hard, bony tumor but the mucous membrane was intact. The tumor thus impinged upon the left lower lobe bronchus (Fig. 2). The main mass of tumor, however, occupied the lateral and posterior portions of the left lower lobe and extended out to the pleura.

The bony mass could be cut by the saw only, and was extremely eburnated. On section, a roughly oval, lobulated mass of firm, gray tissue sharply demarcated from the surrounding lung was observed. A large part of the tumor, which occupied the posterior portion of the left lower lobe, lay subpleurally. This mass measured 11 by 8 cm. (Fig. 3). In places it was of bony firmness, elsewhere it was gray and translucent and varied from cartilaginous to fleshy in consistency. In other areas multiple, fine, bony spicules could be palpated. In the peripheral portion of the large mass a secondary, irregular cavity filled with debris and bony spicules measuring approximately 5×2.5 cm. was encountered.

The tumor extended posterior to the left major bronchus. Here it was lobulated, fleshy in consistency, and at first the mass resembled lymph nodes (*C*, Fig. 2). Section through this area revealed these masses to be continuous with the large mass described above.

In no situation did the tumor mass break into a bronchus or appear to be derived from bronchial wall. Several lymph nodes in the mediastinum showed a mottled gray and black cut surface without gross evidence of tumor invasion.

The major portion of the left upper lobe was gray, firm, and consolidated. Multiple small, irregular cavities with shaggy walls filled with pus were noted. These varied from 2 to 15 mm. in diameter. The portion thought to be left lower lobe, which lay below the tumor mass, was noncrepitant, gray, and fibrous, and contained several irregular, bronchiectatic cavities lined by thick, hyperemic mucous membrane and varying from 1 to 2.2 cm. in diameter.

There was a brain abscess in the left cerebral hemisphere with focal fibrinopurulent meningitis, but the balance of the organs failed to show significant changes.

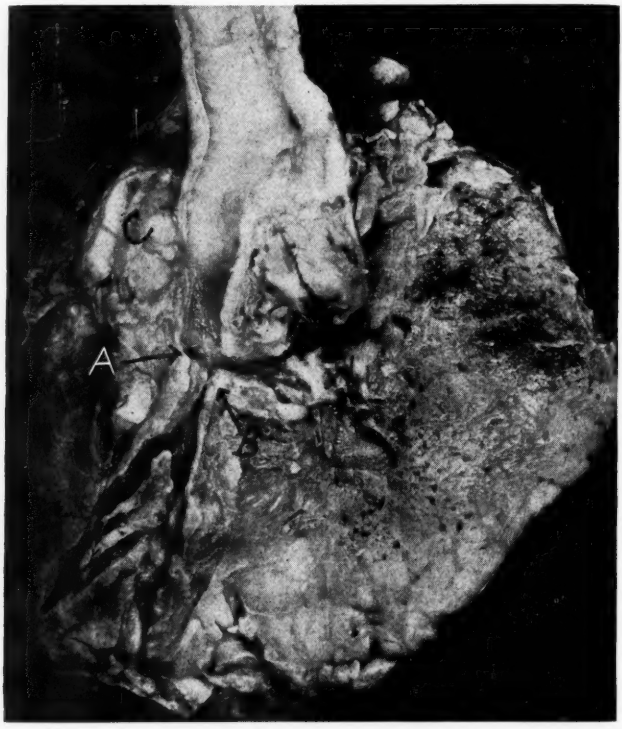

Fig. 2.—The left upper lobe bronchus, *A*, has been narrowed. The lower lobe bronchus, *B*, is slitlike. *C*, Represents a portion of tumor.

Microscopic Findings.—Microscopically, the tumor was quite variable in composition. Sections taken from the softer and more fleshy portions of the tumor showed a richly cellular pattern in which the cells varied from fat-spindle to oval, or round. The nuclei of these cells were vesicular, varied considerably in size, shape, and staining quality, and showed not infrequent bizarre and atypical mitotic figures (Fig. 4). Between the cells there was a variable amount of poorly defined, pink-staining matrix, which at times appeared to be protoplasmic processes of cells and at other times appeared to be amorphous or fibrillar. In some situations numerous giant cells containing from two to twenty nuclei were seen scattered irregularly throughout the tissue. In a few situations some of the tissue appeared to lie in lacunar spaces, suggesting a transition to young cartilage cells.

In other portions of the fleshy tumor the cells appeared more spindlelike. Here the tumor was slightly whorled and richly cellular and occasional mitotic figures were encountered.

Here and there small islands of fibrocartilage were seen (Fig. 5) and the tumor was well vascularized by thin-walled blood vessels, some of which contained hyaline thrombi.

Sections taken through the fleshy tissue in which fine bony spicules were felt showed a richly cellular pattern in which the cells were distinctly spindle-shaped and uniform in size and shape. Here numerous irregular islands of hyaline and fibrocartilage showing various stages of calcification were seen. Some areas were encountered where cells were polymorphic and atypical and near the periphery of one nodule the mediastinal fat was invaded. All types of transition from spindle cells to cartilage were noted.

Fig. 3.—Saggital section through left lung showing tumor with secondary cavity and adjacent nodules. Note the organizing pneumonia and bronchiectasis of the left upper lobe.

Sections taken from the bony mass which required decalcification showed a rather cellular fibrous stroma in which numerous islands of cartilage in various stages of calcification were seen. In some places these calcified masses of cartilage branched and anastomosed to form typical bony trabeculae which contained Haversian canals. No fatty marrow was encountered and no foci of hemotopoiesis were seen. Between the bony trabeculae and in the cellular portion several irregular and elongated spaces were encountered which were lined by single layers of low columnar to cuboidal, ciliated epithelium (Fig. 6). This epithelium rested directly upon the cellular tumor stroma and contained no recognizable muscle or glands (Fig. 7). In the periphery of this section the capsule of the tumor was absent and tumor with bony spicules was found invading the compressed, adjacent lung and surrounding small bronchioles (Fig. 8).

Section through the left upper lobe bronchus where the tumor impinged upon the bronchial wall and narrowed the lumen showed a richly cellular spindle-shaped mass of tumor cells in which spicules of bone were present. The tumor lay adjacent to but did not involve or appear to be derived from the bronchial cartilage or perichondrium. Adjacent lymph nodes showed no metastatic tumor (Fig. 9).

Sections taken to include the secondary cavity within the tumor mass showed extensive bland necrosis of the cellular matrix with only a few bony and calcified cartilaginous trabeculae remaining.

To summarize, the tumor is large and is intrapulmonary in position. It is roughly lobulated, sharply demarcated, and as far as can be determined, does not arise from bronchial wall. This tumor showed every stage of bone genesis from a primitive osteoid tissue passing through cartilage formation to calcification. In one situation there is included deep within the bony portion of the tumor several structures lined, in part, by ciliated epithelium which, in all probability, are integral parts of the tumor rather than residual lung structures surrounded by invading tumor. In many places, from a histologic point of view, the tumor is frankly benign. In other situations malignancy cannot be excluded upon objective histologic grounds. In one area mediastinal fat has been invaded and in another the capsule of the tumor is absent and invasion of lung parenchyma is seen.

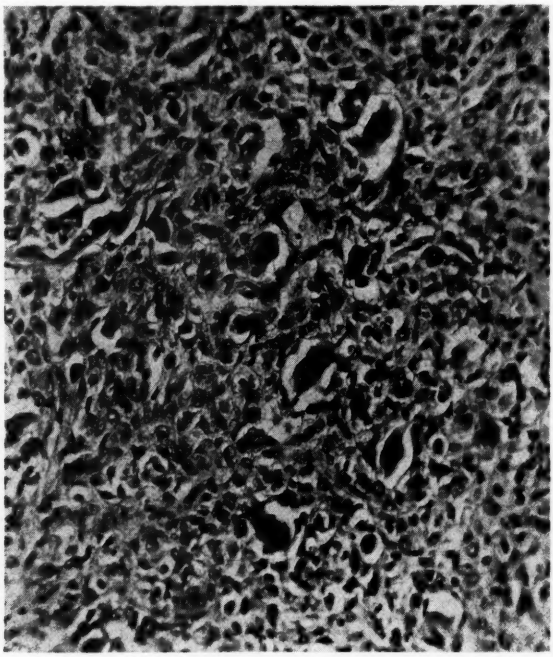

Fig. 4.—Cellular polymorphic area containing numerous giant cells. In this area, mitotic figures were frequent and malignancy could not be excluded upon objective histologic grounds.

DISCUSSION

What is the nature and genesis of this tumor? There is little doubt that this tumor is primary in origin in the lung. The remote possibility that this tumor represents a metastasis from a small, cryptic, primary malignant bone tumor cannot be seriously considered. The absence of other symptoms and signs, the solitary nature of the tumor, its partial encapsulation and lack of metastases elsewhere preclude such a possibility.

This tumor does not appear to be connected with or arise from any existing cartilaginous rings, although the bronchus rides over it. The tumor lies within the lung substance, which it has pushed aside, and largely beneath the pleura. It encroaches upon the hilum. In this respect the present tumor is similar in position to those so-called chondromas of the lung collected and reported by Hicky and Simpson,[3] Verga,[4] Benninghoven and Pierce[5] and McDonald, Harrington, and Clagett.[6] All the above authors have noted the close proximity of these tumors to the pleura. In none of their cases did the tumor appear to arise in the mediastinum and extend secondarily into the lung parenchyma.

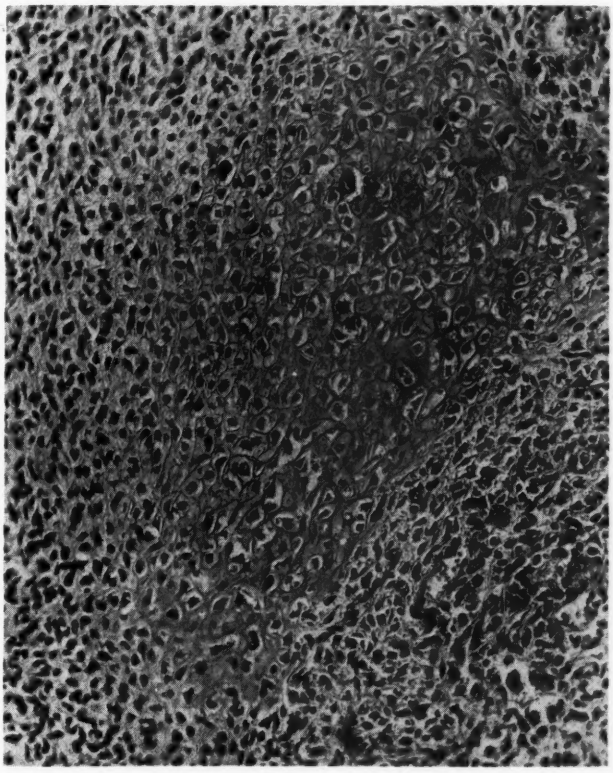

Fig. 5.—Area of fibrocartilage developing in cellular polymorphic portion of tumor.

The fact that the present tumor contains bone and shows transitions from cellular spindle-cell matrix through cartilage, calcification, and ossification, does not necessarily argue its derivation from osseous tissue, whether arising from a focus of osseous metaplasia in a scar, from a healed tuberculous lesion, or from a focus of bony metaplasia in a cartilaginous ring, for the so-called chondromas of the lung which have been reported in the literature have shown various stages of calcification and ossification and, indeed, such changes might

readily be expected. The majority of investigators describe their tumors as having a rocklike consistency. The possibility that this tumor arose on the basis of bony metaplasia which has been described in Henke-Lubarsch[7] as occurring in chronic organizing pneumonia seems unlikely, for these bony spicules are small, needlelike, widely dispersed, and never tumorous in character, and no transitions between cartilage and cellular matrix are observed such as occurred in this tumor.

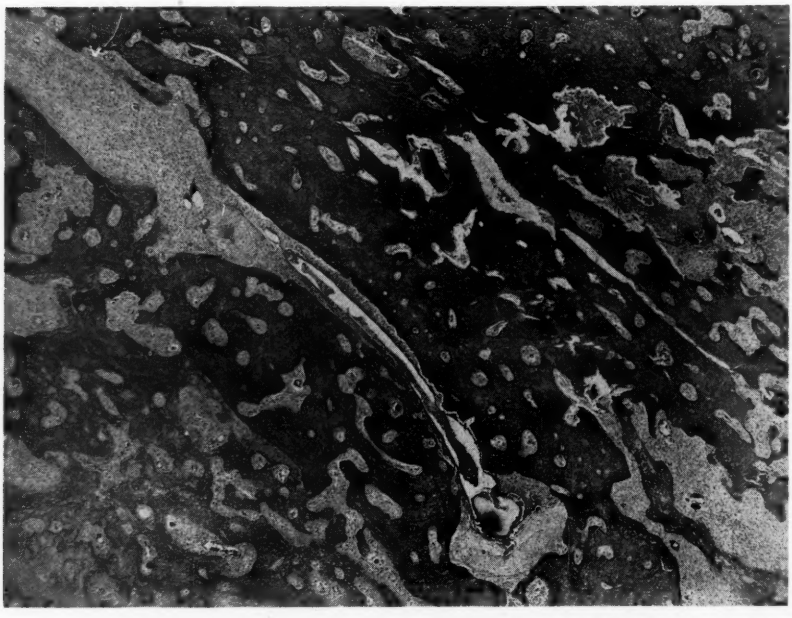

Fig. 6.—Low-power view of frankly bony portion of tumor with osteoid matrix. In the center is a long slender space lined by cuboidal epithelial cells, some of which are ciliated.

The findings of irregular spaces deep within the tumor which are lined by epithelium, some of which is ciliated, are, perhaps, of greatest significance in attempting to arrive at an understanding of the genesis of this tumor. The fact that these spaces are composed only of a layer of epithelial cells resting upon the cellular matrix of the tumor without other recognizable components of bronchial or bronchiolar wall indicates that these structures are an integral part of the tumor rather than remnants of bronchial structures which were surrounded and isolated by invading tumor.

In the great majority of the so-called chondromas of the lung reported in the literature, various admixtures of glandular spaces, lined at times by ciliated and nonciliated epithelium with or without papillary infolding, fat, blood vessels, smooth muscle, and occasionally lymphocytes, have been noted. Various names such as "adeno-fibro-lipo-chondromyxomatosum" "lipo-chondro-adenoma," "ad-

eno-chondroma" and "lipo-chondroma" have been applied to these tumors. Bayer[8] reported five cases of what he called bronchial hamartoma and Goldsworthy[2] reported a case which he labeled hamartoma chondromatosum pulmonis.

The presence, therefore, of both mesenchymal and ectodermal elements in the above-mentioned and in the presently discussed tumor suggests that we are dealing with a hamartoma or teratoma. According to Albrecht,[1] who first used the term, a "hamartoma (from the Greek, meaning to fail or err) is a tumorlike malformation in which occurs actually only an abnormal mixing of the normal components of the organ. The abnormality may take the form of a

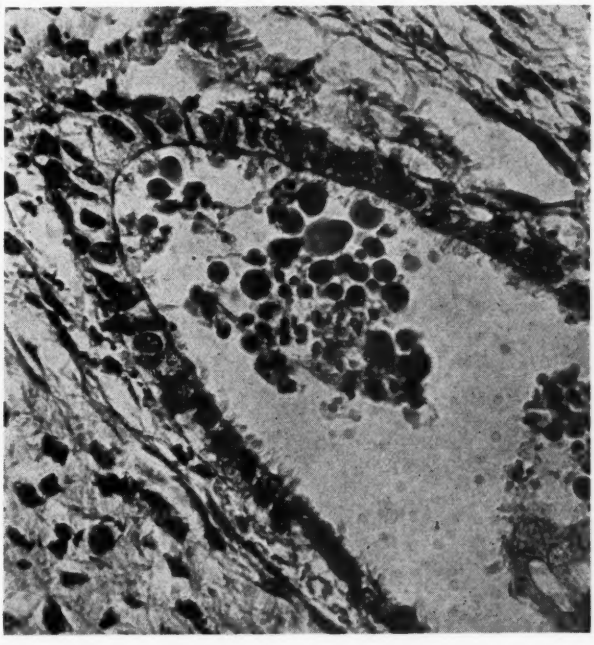

Fig. 7.—High-power view of space lined by ciliated epithelium. Note absence of muscle, glands, and note that the epithelial cells rest directly upon osteoid matrix.

change in quantity, arrangement, or degree of differentiation, or may comprise all three. The deduction to be drawn from the histologic examination of these formations is that they have originated in an abnormal mixing of the elements or from disturbance of their development."

In the tumor, in our case, the presence of cartilage and of spaces lined by ciliated epithelium can be regarded as an abnormal mixing of normal components of the bronchus. The quantities of these elements and their degree of differentiation do not invalidate the definition. Indeed, Borst,[6] suggests that such developmental disturbances may become the seat of growth excesses and may become truly neoplastic or even malignant in character. Borst[9] also noted

that cartilage occurs in a large proportion of hamartomas and suggests that the cartilage present in many of the chondromas of internal organs was due, in part, to metaplasia of connective tissues and, in part, to embryonic scattering of cartilaginous germ centers. Mallory[10] has also stressed the importance of metaplasia in cartilage-containing tumors of soft parts. In the present case, areas were encountered in which dedifferentiation was quite marked and where the tumor was definitely neoplastic if not malignant in character.

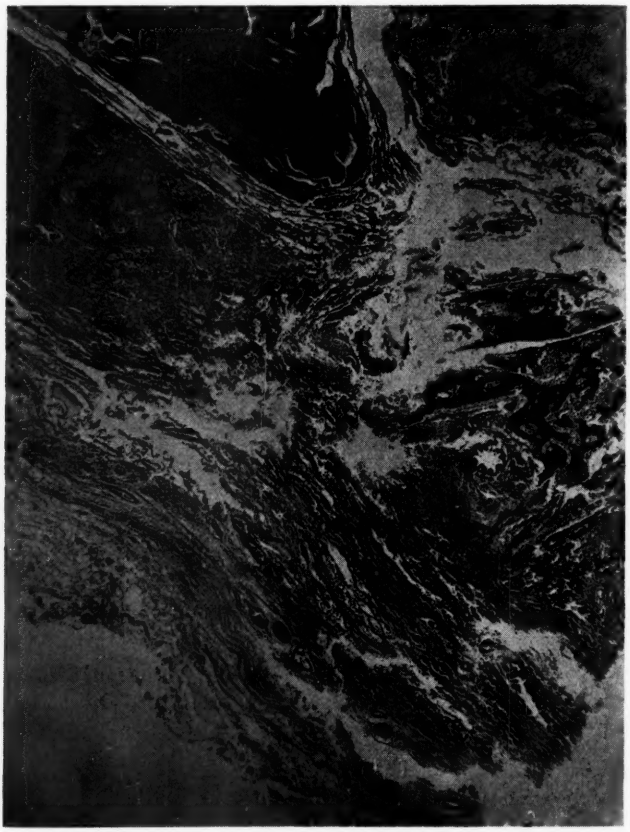

Fig. 8.—Compressed peripheral portion of lung (lower left) showing lack of capsule of tumor. Note that the tumor surrounds an intact bronchiole on the right.

The term hamartoma is all-inclusive and does not necessarily refer to a given organ or tissue overgrowth type. Therefore, with Goldsworthy,[2] who suggested the name of "hamartoma chondromatosum pulmonis," the authors would agree that specific localization and description might be included in the term and, to this end, one might call the present tumor "hamartoma osteochondromatosum pulmonis malignum."

There are those who may possibly wish to employ the term "mixed tumor" in the sense of Womack and Graham.[11] For those who wish to regard this tumor as a teratoma in which there has been an overgrowth of one element with suppression of the others, the following objections may be raised. No other structures than those normally found in this region were discovered in this tumor. The present tumor, unlike the majority of teratomas, does not arise anatomically from the mediastinum.

Fig. 9.—Left upper lobe bronchus passing over tumor. Note cellular structure of tumor, left, containing bony spicules. The lymph node on the right is not invaded. Note intact perichondrium about bronchial cartilages.

Although, from a clinical point of view and in the absence of metastases, this tumor may be regarded as benign, it is apparent from an objective histologic point of view that malignancy cannot be excluded. Furthermore, evidence of lack of capsule and local invasion in the periphery of the tumor is present. This recalls the unique case of a pulmonary osteochondrosarcoma reported by Greenspan[12] which showed locally invasive features. It is probable that the origin of his reported tumor was from a hamartoma, although no epithelial-lined structures or other tissue types were noted. From a purely

theoretical point of view, there is no valid reason why a hamartoma or any other congenital malformation may not, under certain conditions, show malignant changes.

From a clinical point of view our case is of interest in that it may be assumed that the tumor had been present for many years. When it impinged upon the narrowed major bronchi it produced signs not unlike those associated with bronchial obstruction due to carcinoma. The original and all subsequent x-rays were suggestive of tumor but failed to reveal the characteristic findings noted in the x-ray of the specimen. Most so-called pulmonary chondromas are silent because of their usually small size and intraparenchymal position. In Klages'[13] case, the first in which the diagnosis was made roentgenologically and confirmed by histological examination of the specimen, the patient was free of symptoms. Similarly, in one of the two cases reported by Benninghoven and Pierce,[5] which was treated surgically, no symptoms were present. In only one of the three cases reported by McDonald, Harrington and Clagett[6] in which surgical removal was performed, were symptoms present, and these consisted of dyspnea on exertion and a burning sensation in the anterior portion of the thorax. It is to be noted that intralumenal polypoid tumors which may contain chondromatous elements such as those reported by Moore,[14] Gebauer,[15] and Davidson[16] are not included with hamartomas.

So-called pulmonary chondromas are not unique in man but have been noted in domesticated animals.[17]

SUMMARY

An unusually large hamartoma (so-called chondroma) of the lung is reported. It was responsible for signs suggestive of bronchiogenic carcinoma. It would perhaps be preferable to describe the hamartoma in our case as a hamartoma osteochondromatosum pulmonis malignum. The tumor which we have described showed features which upon objective grounds do not permit one to exclude the possibility of malignancy.

REFERENCES

1. Albrecht, E.: Ueber Hamartome, Verhandl. d. deutsch. path. Gesellsch. **7**: 153-157, 1904.
2. Goldsworthy, N. E.: Chondroma of the Lung (Hamartoma Chondromatosum Pulmonis): With Report of a Case, J. Path. & Bact. **39**: 291-298, 1934.
3. Hicky, P. M., and Simpson, W. M.: Primary Chondroma of the Lung, Acta radio. **5**: 475-500, 1926.
4. Verga, Pietro: I condromi del pulmona, Pathologica **24**: 1-22, 1932.
5. Benninghoven, Carl D., and Pierce, Carleton B.: Primary Chondroma of the Lung, Am. J. Roentgenol. **29**: 805-812, 1933.
6. McDonald, John R., Harrington, Stuart W., and Clagett, O. Theron: Hamartoma (Often Called Chondroma) of the Lung, J. THORACIC SURG. **14**: 128-143, 1945.
7. Henke, F., and Lubarsch, O.: Handbuch der Spegiellen Pathologischen Anatomie und Histologie, Berlin, 1931, Julius Springer, vol. 3, part 3, p. 516-517.
8. Bayer, R.: Beitrag zur kenntnis der sog. Lungenchondrom, Virchow's Archiv. **274**: 350-353, 1929-30.
9. Borst, M.: L. Aschoff's Pathologische Anatomie, ed. 7, Jena, 1928, vol. 1, pp. 689 and 726.
10. Mallory, T. B.: Groups of Metaplastic and Neoplastic Bone and Cartilage Containing Tumors of the Soft Parts, Am. J. Path. **9**: 765, 1933 (suppl.).

11. Womack, Nathan A., and Graham, Evarts A.: Mixed Tumors of the Lung, Arch. Path. **26**: 165-206, 1938.
12. Greenspan, E. B.: Primary Osteoid Chondrosarcoma of the Lung: Report of a Case, Am. J. Cancer **18**: 603-609, 1933.
13. Klages, F.: Über die Chondrome der Lunge, Beitr. z. klin. Chirurg. **151**: 661-671, 1931.
14. Moore, R. A.: Ueber ein polyposes Chondrom des Bronchus, Centralbl. f. allg. Path. u. path. Anat. **55**: 321-324, 1932.
15. Gebauer, P. W.: A Case of Osteochondroma of the Bronchus, Ohio State M. J. **34**: 398-399, 1938.
16. Davidson, M.: A Case of Primary Chondroma of the Bronchus, Brit. J. Surg. **28**: 571-574, 1941.
17. Casper, M.: Geschwulste der Tiere, Ergebn. d. allg. Path. u. path. Anat. **3**: 692-716, 1897.

ADENOMAS ARISING FROM SMALL BRONCHI NOT VISIBLE BRONCHOSCOPICALLY

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BRONCHIAL adenomas usually occur in the larger bronchi and can be visualized through the bronchoscope. The diagnosis is often established by the microscopic examination of biopsy material removed endoscopically. In an extensive discussion of bronchial adenoma by Foster-Carter[1] in 1941, the statement is made: "A bronchial adenoma arises invariably in one of the larger bronchi and there is no record of this tumor occurring in the periphery of the lung." Apparently all of the adenomas reported in the literature at that time were in portions of the tracheobronchial tree that could be visualized through the bronchoscope. Moersch and associates[2] recently stated, "Adenomas invariably arise from the main stem or larger bronchi." Among the patients with bronchial adenoma that we have treated, there are five in whom the tumor was located in a branch bronchus not accessible to the bronchoscope. In each instance the tumor was large enough to be clearly demonstrable on the roentgenogram of the chest. In two patients an x-ray was taken because of thoracic symptoms. In two other cases the tumor was found during an x-ray survey. In one patient the pulmonary lesion was found during a gastrointestinal roentgen study. Since the tumor did not arise from a large bronchus in any of the cases, it is understandable that none of the patients had any history or symptoms of secondary pulmonary suppuration. In four of the cases the tumor was located near the hilar area (Figs. 1, 2, 3, and 4) but arose from a small branch bronchus rather than from the main lobar bronchus. In the fifth case the tumor was at the periphery of the lung close to the diaphragm (Fig. 5). Three of the patients were asymptomatic, one patient had had several hemoptyses, and one had slight pain in the chest. Lobectomy was done in four cases and partial lobectomy performed for the peripheral tumor in the fifth case. All patients had an uncomplicated postoperative course and are in good health at this time, two years eight months to six months after operation.

In the four cases in which the bronchial adenoma was located near the hilar portion of the lung, a correct diagnosis was made at the time of operation on the basis of the gross characteristics of the tumor. Although the peripherally located tumor had a similar consistency, its most unusual location made us hesitate to diagnose an adenoma until that diagnosis was demonstrated by pathological examination (Fig. 6).

It is important that the thoracic surgeon recognize the gross characteristics of bronchial adenoma. The tumor is moderately soft, being considerably softer than the typical carcinoma. Not infrequently some lobulation is present. The

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tumor is usually reddish-brown in color. If a portion of the tumor projects into the mediastinum, small veins are often seen crossing over the surface. In most instances a portion of the tumor can be enucleated from the adjacent pulmonary tissue or mediastinal tissues, but there is no cleavage plane in the region of the bronchus of origin. If the tumor projects into an interlobar fissure, it may be noted that when the pleura is divided over the tumor, that portion of the tumor which projects toward the adjacent lobe can be readily freed, so that lobectomy is feasible. We do not feel that pneumonectomy is necessary in cases of bronchial adenoma that are typical and so located that the tumor can be completely removed by lobectomy. None of our patients had involvement of the regional lymph nodes.

CASE REPORTS

CASE 1.—G. H., male, aged 40, was admitted to Memorial Hospital on Dec. 6, 1943. A year previously the patient noticed slight dyspnea on exertion and fatiguability. Several hemoptyses had occurred after exertion. Physical examination was negative. Roentgenogram of the chest revealed an elongated, well-demarcated shadow extending out from the left hilum. Associated with this mass there was a moderate increase in density of the anteromedial portion of the left upper lobe suggesting a partial atelectasis. The remaining lung fields were clear. The roentgenographic findings indicated a tumor at the left hilum, probably arising in the region of the ventral segment of the left upper lobe (Fig. 1). Bronchoscopy was negative.

At operation on Dec. 7, 1943, a well-encapsulated ovoid lesion which lay in close proximity to the main stem bronchus and hilum was found. A dissection type of left upper lobe lobectomy was performed. On examination of the surgical specimen, a small portion of the lobe was found to be dark in color, moderately firm in consistency, and apparently airless; the remainder was aerated. Involving the hilar region and situated just adjacent to the main lobar bronchus was an irregular, ovoid-shaped tumor. It was pink in color, showed some lobulation, and was apparently encapsulated and well demarcated from the adjacent pulmonary tissue. It measured 4.5 × 3.1 × 2.3 cm. Section of the lung showed that there were three main branches of the lobar bronchus. Two were situated below the tumor and the uppermost one of these two was displaced downward and was closely associated with the tumor, but there was a definite line of cleavage between the two. The other branch of the bronchus was located above and lateral to the tumor. Microscopic diagnosis was bronchial adenoma. The patient had an uneventful postoperative course and has been well and asymptomatic since.

CASE 2.—K. I., female, aged 27, was admitted to Lenox Hill Hospital on June 26, 1944. Five years prior to admission the patient developed pain in the chest and an x-ray revealed a mass in the left lung. Repeated roentgenograms since then had shown gradual enlargement of the mass, although the patient had no pulmonary symptoms excepting occasional slight discomfort in the chest. On admission a chest roentgenogram revealed an oval, sharply marginated shadow of increased density at the posterior aspect of the lower portion of the left hilum, measuring about 4 cm. in diameter (Fig. 2). On June 28, 1944, a thoracotomy was performed. In the upper part of the lower lobe, bulging somewhat into the interlobar fissure, a rounded, slightly firm tumor mass was found in close association with the bronchus of the lower lobe. The mass was slightly lobulated and could be enucleated from the adjacent pulmonary tissues over a considerable area, except at its attachment to the bronchus. The lower lobe was air-containing and showed no atelectasis or signs of inflammation. A left lower lobe lobectomy of the dissection type was performed. Examination of the surgical specimen revealed that the tumor arose from a small branch bronchus adjacent to the lobar bronchus. No inflammation was present. The pathological diagnosis was bronchial adenoma. The postoperative course was uneventful and the patient has remained well.

CASE 3.—C. K., male, aged 42, was admitted to Lenox Hill Hospital on Feb. 1, 1945. One month prior to admission a routine x-ray of the chest at his place of employment showed

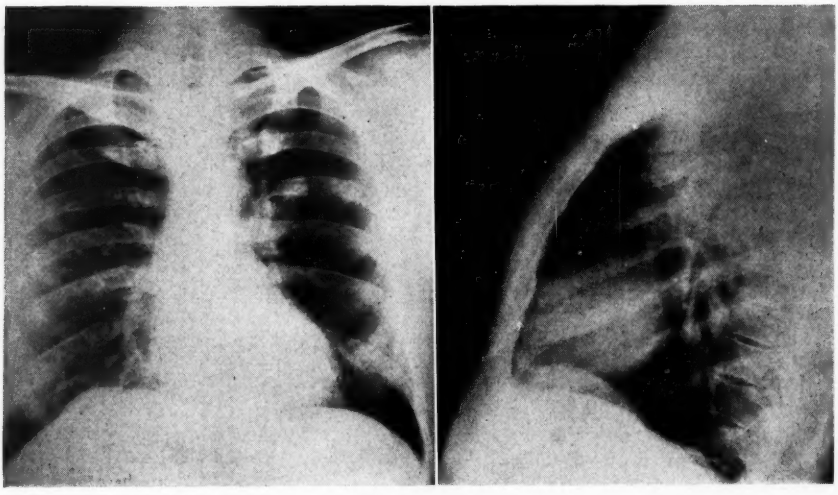

Fig. 1.—Mass in hilar region of left upper lobe.

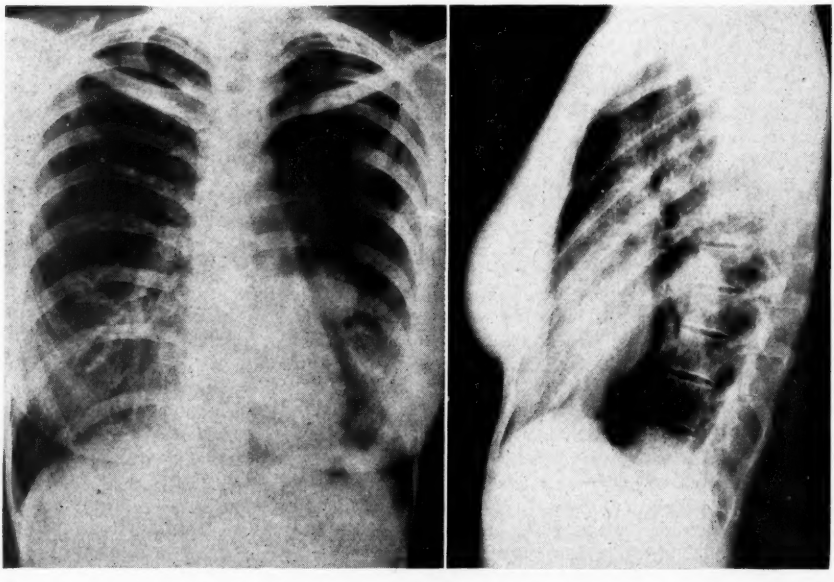

Fig. 2.—Circumscribed mass near hilum of left lower lobe.

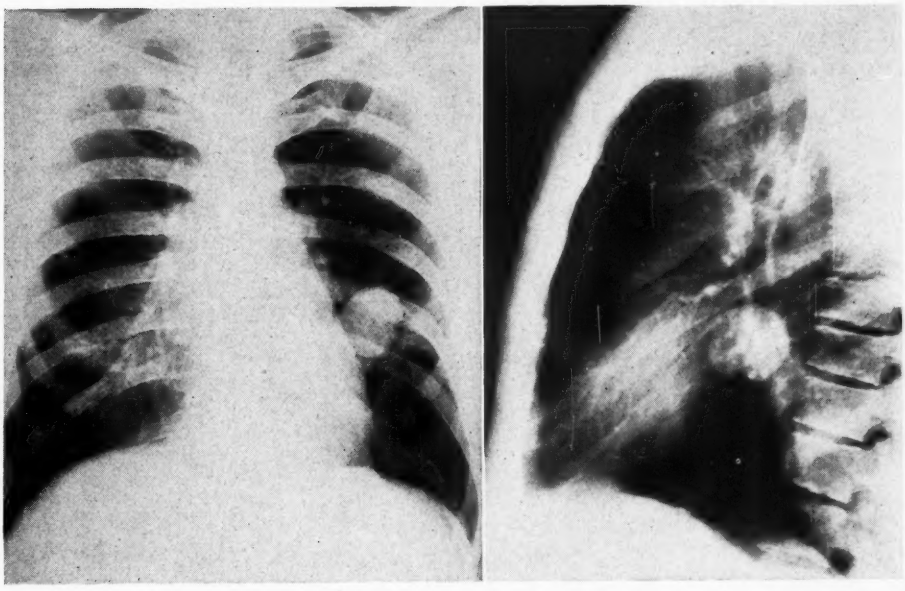

Fig. 3.—Sharply circumscribed mass near hilum of left lower lobe.

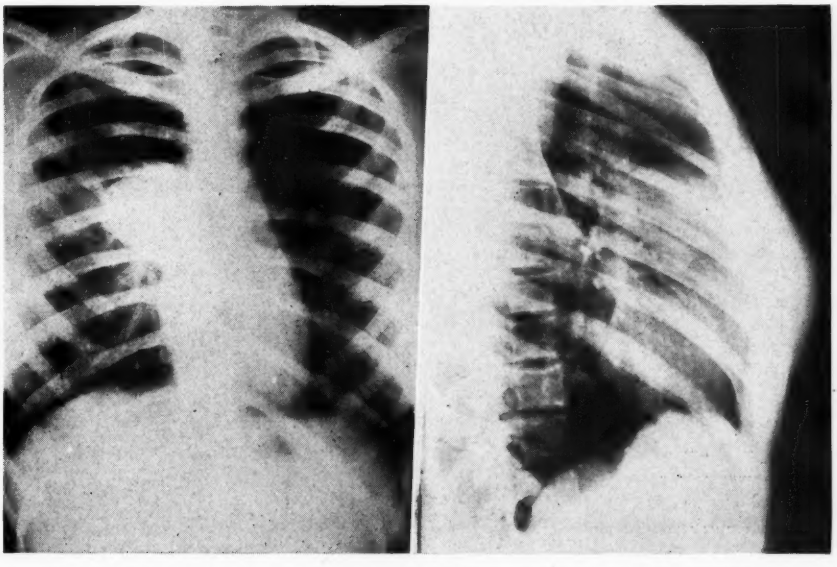

Fig. 4.—Large mass in anterior portion of right upper lobe with distal atelectasis.

a lesion of the left lung. He had had no symptoms. No previous chest x-ray had ever been taken. Physical examination was negative. Roentgenogram of the chest showed a globular shadow of increased density, measuring about 4 cm. in diameter, located in the left lower lobe adjacent to the inferior portion of the hilum (Fig. 3). At operation on Feb. 3, 1945, a mass could be palpated in the hilar portion of the left lower lobe. The tumor bulged into the interlobar fissure and encroached slightly on the upper lobe. On exposure the mass was somewhat lobulated, and a portion of it could be enucleated from the surrounding tissue. A dissection type of left lower lobe lobectomy with complete removal of the tumor was done. Examination of the gross specimen revealed that the tumor did not present in the lumen of the main lobar bronchus and there was no obstruction of this bronchus. The tumor appeared to arise from a small branch bronchus adjacent to the main bronchus. No inflammation was present. Microscopic examination of the tumor revealed a bronchial adenoma. The postoperative course was uneventful and the patient has remained well.

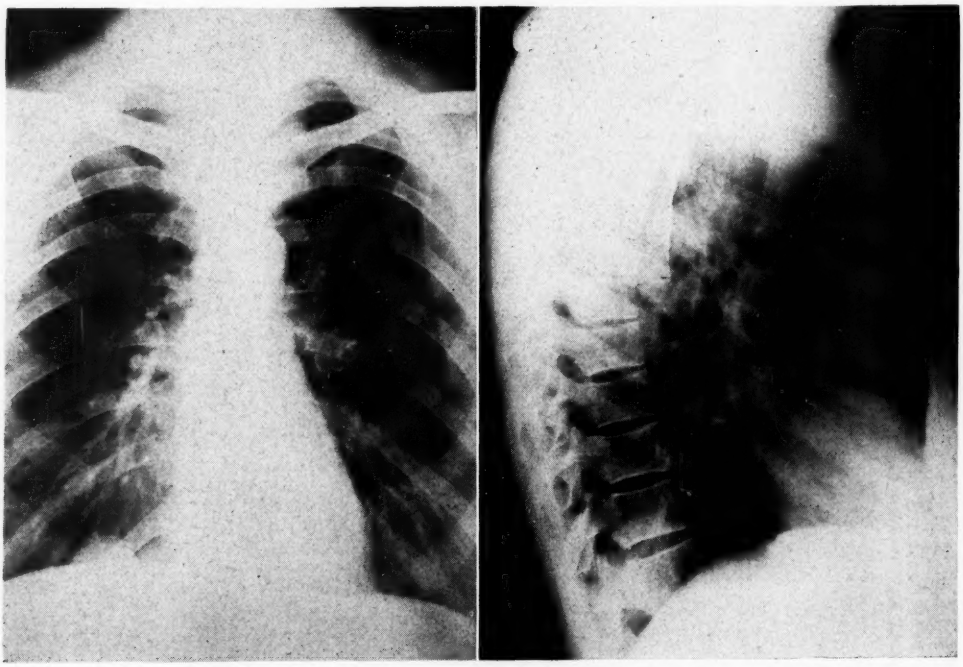

Fig. 5.—Posteroanterior film shows rounded mass projecting partly above dome of right diaphragm. Lateral film shows mass posteriorly in costovertebral portion of right lower lobe just above diaphragm.

CASE 4.—I. D., Negress, aged 16, was admitted to Lenox Hill Hospital on Jan. 30, 1946. Routine roentgenogram of chest made in a tuberculosis survey in November, 1945, revealed a mass in the right middle lung field. The patient had always been in good health and had had no symptoms referable to the lungs. Physical examination was negative. Roentgenogram of the chest showed an irregularly shaped mass measuring approximately 6 cm. in its greatest dimension, situated near the root of the right lung. The lateral film localized the mass anterior to the hilum in the region of the short fissure. A small area of slightly increased density was noted in the lung field lateral to the tumor mass (Fig. 4). Bronchoscopy was negative. Thoracotomy was performed on Feb. 1, 1946. There were vascular adhesions pres-

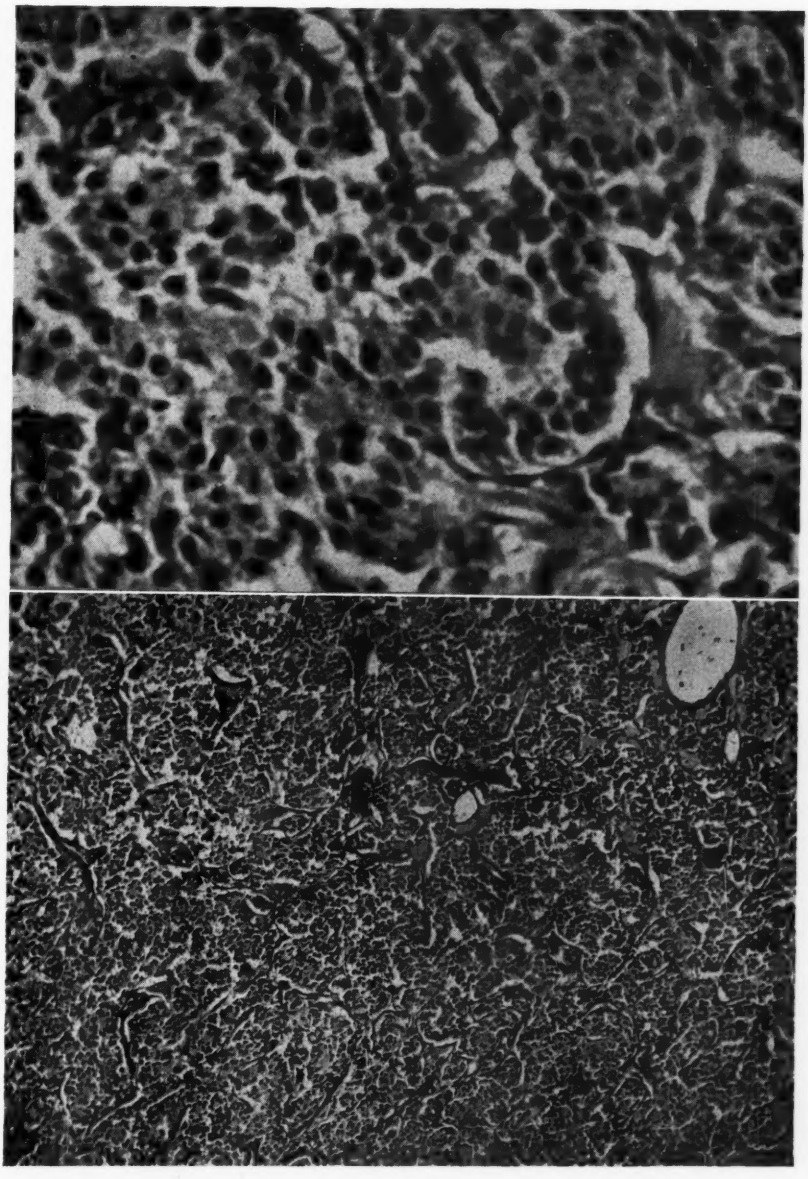

Fig. 6.—Photomicrographs of the peripherally located bronchial adenoma.

ent over the lower anterior portion of the upper lobe. This portion of the lobe was airless and had a yellowish appearance. A tumor was present near the hilum of the upper lobe. A dissection type of right upper lobe lobectomy was performed. Examination of the specimen revealed a moderately firm mass arising from the lower anterior branch bronchus. The lung peripheral to the tumor was largely airless and rubbery in consistency, and on section revealed a soft, golden-pink tissue. Microscopic examination of the tumor revealed a bronchial adenoma. Section through the golden-pink pulmonary tissue distal to the adenoma revealed large foam cells with extensive cholesterin deposits. Sections of the lymph nodes showed no tumor. The postoperative course was uneventful and the patient has remained well.

CASE 5.—M. U., female, aged 38, was first seen at Memorial Hospital in January, 1945, when she complained of epigastric pain and postprandial vomiting of seven months' duration. The vomitus had been bloody on two occasions. She had been unsuccessfully treated by diet. Roentgenograms were interpreted as showing a soft tissue mass in the lower peripheral portion of the right lower lobe (Fig. 5) and pyloric obstruction, possibly due to a peptic ulcer. An aspiration biopsy of the lung lesion was reported as probable malignant tumor. At operation on March 8, 1945, a soft, sharply circumscribed tumor mass was found in the right lower lobe just beneath the diaphragmatic and posterior pleural surfaces of the lobe. There was no atelectasis and the adjacent pulmonary tissue appeared normal. Because of the peripheral location of the tumor and the absence of any other pathology in the lobe, a wedge resection of that portion of the lung in which the mass was situated was performed, and frozen section done immediately. The microscopic diagnosis was bronchial adenoma (Fig. 6). On examination of the gross specimen, the tumor mass, measuring $5 \times 5 \times 4$ cm. was found well circumscribed and demarcated from the surrounding pulmonary parenchyma by a fine but definite capsule. In no place did it invade or ulcerate the overlying visceral pleura. The gross appearance resembled lymphoid tissue. The patient had an uneventful postoperative course. Four months later gastric resection was performed for obstructing duodenal ulcer. The patient has been well since that time.

SUMMARY

Five cases of bronchial adenoma arising from small bronchi are reported. In each instance the adenoma was located in a portion of the bronchial tree that cannot be visualized bronchoscopically. Hemoptysis was present in only one case. At the time of operation, none of the patients had any pulmonary suppuration. Lobectomy was performed on four patients. Due to the peripheral location of the adenoma in the fifth patient, a partial lobectomy was done. All patients had an uncomplicated postoperative recovery and are well at this time.

REFERENCES

1. Foster-Carter, A. F.: Bronchial Adenoma, Quart. J. Med. **10**: 139, 1941.
2. Moersch, H. J., Tinney, W. S., and McDonald, J. R.: Adenoma of the Bronchus, Surg. Gynec. & Obst. **81**: 551, 1945.

FASCIA LATA GRAFTS FOR CHEST WALL DEFECTS

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CHEST wall tumors comprise a group of neoplasms which has been adequately covered in the medical literature. Hedblom,[1, 2] in the year 1921, and again in 1933, reviewed all reported cases and added his personal observations. Sommer and Major[3] reviewed all references to the subject from 1933 to 1940 and added fifteen cases of their own. In these reports the authors agreed that the closure of large, surgical, chest defects was sometimes very difficult. Hedblom used various measures for producing adhesions between the lung and that part of the chest wall which was to be resected. His operations were carried out in stages. Iodoform gauze was first packed subcostally for a time before the pleura was opened or the lung itself was sutured to the chest wall. When approximation of the intercostal structures was not possible, Sommer and Major attempted airtight chest wall closure by careful suture of the extracostal muscles, the subcutaneous tissue, and skin. Effler and Blades[4] in a recent discussion of "Thoracic Centers in World War II," also stated that serious defects in the chest wall were not always readily closed with skin, muscle, and bone of the thorax. In a few isolated instances, tantalum plates have been used to bridge the defects in the chest wall, but these endeavors were commonly disastrous because the plate acted as a foreign body and either sloughed out or became loose and moved with respirations and had to be removed.

Fascia lata transplants have been or could possibly be used to serve many different purposes. Kirschner[5] in 1914 listed, among other uses for fascia lata grafts, the possibility of closing defects in the thoracic wall. In 1924, Brenner[6] used fascia lata in the reconstruction of a portion of the male urethra. In 1924, Gallie and LeMesurier[7] used free transplants of fascia lata as living sutures in the treatment of hernia. During World War II, fascia lata transplants were often used in repairing large dural defects,[8] to stop the loss of cerebrospinal fluid and prevent the entrance of organisms through the meningeal defect. In 1930, Zinninger[9] reviewed tumors of the chest wall at the Peiping Union Medical College Hospital. He presented one case of a 23-year-old Chinese carpenter who had a fibrosarcoma of the right epigastrium. It was necessary to excise the entire thickness of the abdominal wall in order to remove the tumor and the peritoneal cavity had to be opened. In closing the wound it was possible to approximate the skin and subcutaneous tissues, but there remained a defect in the peritoneum, muscle, and fascia about the size of a man's hand. This he closed with a layer of fascia lata from the right thigh, fixing it with interrupted silk sutures to the peritoneum and fascia on both sides. The skin flaps were then closed over the fascial transplant.

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It is the purpose of this paper to present four cases of large chest wall tumors in young patients which were resected, and the resultant surgical defects successfully closed with the aid of large, wide, free, fascia lata grafts. The procedure seems to be a practical one, which probably can be used more frequently in the future.

TECHNIQUE

Endotracheal gas, oxygen, and ether anesthesia is employed and the patient is prepared as for any other major chest operation, but, in addition, one thigh is also prepared and draped. After a wide resection of the tumor-bearing portion of the chest wall, a fascia lata graft, the same size as the defect, is taken from the selected thigh (Fig. 1), and the "shiny side," that is, the surface adjacent to the subcutaneous tissue, is placed so as to be in contact with pleura. It is then secured to the parietal pleura and periosteum with multiple interrupted silk sutures as shown in the diagram in Fig. 2. A #18 French catheter is left in the pleural space until the chest is completely closed in order to facilitate the removal of all free pleural air as the anesthetist establishes complete lung expansion. It is important postoperatively to keep the lung completely expanded so that the graft is continuously pressed against its bed at all times.

CASE REPORTS

CASE 1.—M. L., an unmarried 24-year-old native Dutch West Indian woman, first came to Memorial Hospital on Nov. 9, 1945. She stated that in 1941, two weeks after a slight trauma to her left anterior chest well, she had noted a tumor mass in that area. It gradually increased in size and was intermittently painful. The following year it was excised, and the surgeon stated that it was a benign tumor originating in the pectoralis muscle. The mass soon recurred and was excised again in 1944. No report of the second operation could be obtained. Three months later the mass again recurred and the patient was finally referred to the United States for further treatment.

On admission physical examination revealed a well-developed, thin, Negro female in good general condition. The left anterior chest revealed a tumor mass measuring 5 x 5 cm. overlying the second and third ribs. This seemed to be attached firmly to the underlying structures. Below the mass there were two healed surgical scars, and beneath these scars there was another mass measuring 4 x 5 cm. Roentgenograms of the chest showed no evidence of metastasis to the lungs or destruction of ribs.

The patient was admitted to Memorial Hospital and on Dec. 14, 1945, from a biopsy of the tumor, the pathological report was spindle-cell sarcoma. On Jan. 5, 1946, an attempt was made to remove the growth. The subcutaneous tissue overlying the mass, the pectoralis major and minor muscles, and portions of the third and fourth ribs were removed with the tumor. The tumor was seen to invade pleura and no attempt was made to resect this pleural extension of disease.

Convalescence from the first surgical procedure was uneventful and the patient was readmitted to the hospital and operated upon on April 10, 1946. At this time, the left breast was raised as a flap from the chest wall, exposing the bony chest cage. The remaining tumor was excised, together with portions of the second, third, fourth, and fifth ribs, the intervening muscular bundles, and the underlying parietal pleura (Fig. 3), leaving a chest wall defect measuring 15 x 12 cm. A large graft of fascia lata was then removed from the right thigh and sutured into this defect. The lateral margin of it was sutured to the pleura and periosteum, and the medial margin was attached to the pleura, pericardium, and other

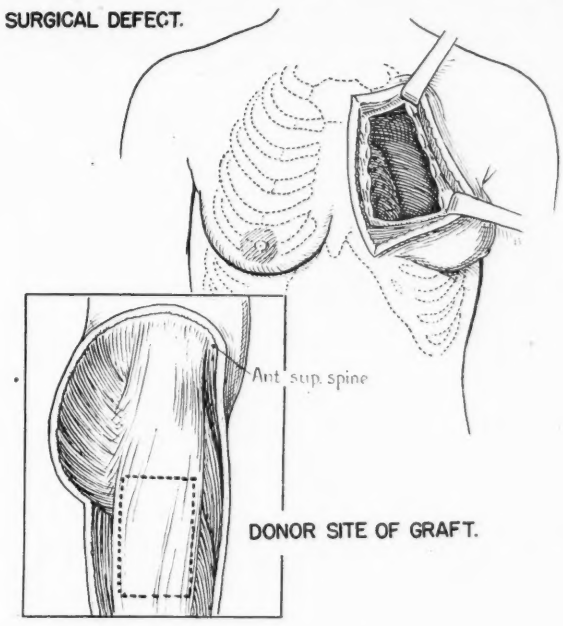

Fig. 1.—Surgical defect.

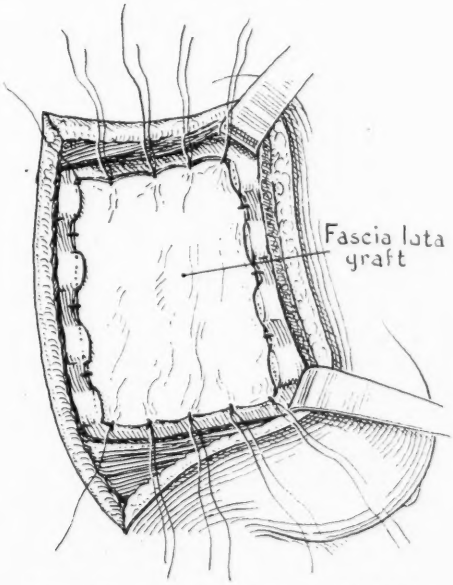

Fig. 2.—Free graft sutured in surgical defect.

soft tissues and muscles as they presented themselves. The skin flap and breast were then sutured in place over the fascia lata graft. Care was taken to fully inflate the lung before the operation was completed.

The patient tolerated the procedure very well, and the postoperative convalescence was without incident (Fig. 4). The lung remained well expanded, and there was no pleural effusion. Repeat postoperative x-rays showed no evidence of pleural reaction (Fig. 5).

The postoperative pathologic diagnosis was low-grade spindle-cell sarcoma.

CASE 2.—J. L., a 55-year-old white, unmarried female, entered Memorial Hospital Aug. 6, 1935, complaining of a lump in the left side of her abdomen which had been present, but symptomless, for two years. For several months prior to admission the mass had increased noticeably in size.

Physical examination was negative except for the tumor mass, which measured 12 x 5.5 cm. was elongated in shape, and seemed to originate near the left costal margin in the region of the anterior axillary line. The mass was elastic in consistency, firmly attached to the underlying bony structures, but not to the overlying skin. Roentgenograms of the chest showed considerable destruction of the left tenth and eleventh ribs.

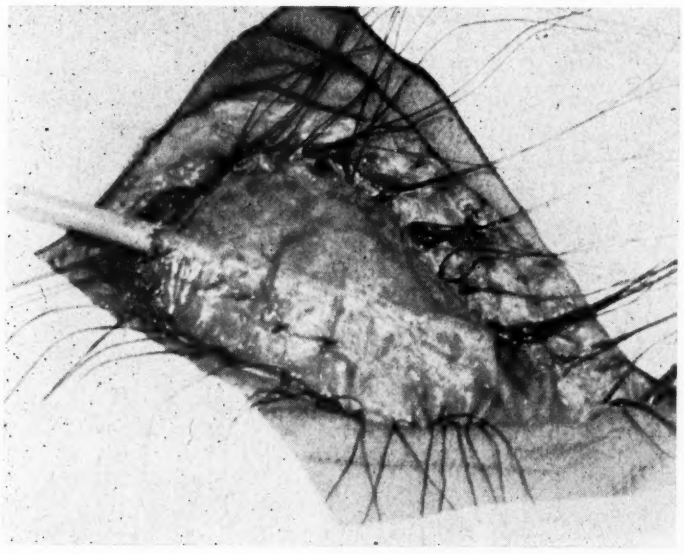

Fig. 3.—Graft in surgical defect.

The mass was aspirated and pathologic examination revealed it to be a chondromyxosarcoma. In preparation for surgery an artificial pneumothorax was established on the left side. On Aug. 16, 1935, operation was performed and a bulky tumor, measuring about 15 cm. in diameter was found to involve the anterior midportions of the left ninth and tenth ribs. It was adherent to the diaphragm and to peritoneum and could be palpated externally where it invaded the muscles of the anterior upper abdominal wall. Because of the adherence of the tumor, portions of the peritoneum, pleura, and diaphragm had to be excised, together with sections of the ninth, tenth, eleventh, and twelfth ribs (Fig. 6 *A* and 6 *B*). A long, wide strip of fascia lata was obtained from the left thigh and sutured to the diaphragm and pleura. The fascia lata graft actually substituted for a part of the diaphragm. The muscle tissue could not be approximated; the skin and subcutaneous tissues were sutured over the graft.

Postoperatively, the patient developed an effusion in the left pleural cavity and carried a few degrees of fever, but the wound healed well and sutures were removed by the eighth day. On the eighteenth postoperative day the patient suddenly went into shock and expired. An autopsy was obtained. The cause of death was found to be a thrombophlebitis of the left internal iliac vein with metastatic, bilateral, pulmonary emboli. Examination of the left pleural cavity showed an accumulation of a small amount of serosanguineous fluid. The graft had taken well to the diaphragm and pleura. There was no adherence of the graft to the visceral pleura.

The final pathologic diagnosis was low-grade chondrosarcoma.

CASE 3.—E. M., aged 21, entered the United States Naval Hospital, Brooklyn, N. Y., Oct. 23, 1944, because of a lump on the right chest. He stated that without known cause, a small mass had appeared on his right chest posteriorly and slowly increased in size, but caused him no pain or disability. An operation under local anesthesia at Quonset Point, R. I., Oct. 16, 1944, revealed the tumor, which was incompletely removed.

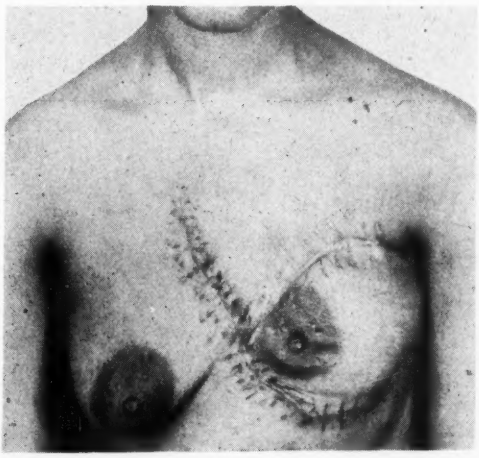

Fig. 4.—Postoperative appearance.

Physical examination revealed a recent scar 10 cm. in length over the right ninth rib posteriorly. The remainder of the physical examination was essentially negative.

On Nov. 29, 1944, a second operation was carried out and portions of the seventh, eighth, and ninth ribs resected on the right side with a block removal of the tumor and that portion of the chest wall. The resulting defect was closed with a broad sheet of fascia lata measuring 15 x 10 cm. 100,000 units of penicillin and 2 Gm. of sulfanilimide powder were placed in the wound. The tumor measured 18 x 16 cm., it was lobulated and movable except at its base, where it was seen to arise from the chest wall and ribs, and at its apex, where it was intimately adherent to the lung. The chest was closed without drainage. On Dec. 5, 1944, thoracentesis, right chest, yielded 700 c.c. Burgundy-colored fluid. On each of the following two days he was given 500 c.c. of whole blood. On Dec. 7, 1944, 1,050 c.c. of clear Burgundy-colored fluid was removed from the right pleural cavity. On Dec. 16, 1944, an x-ray showed the presence of a slight pneumothorax. Repeat x-rays of the chest showed a gradual disappearance of the pneumothorax and pleural effusion.

The postoperative pathologic diagnosis was endothelioma of pleura.

CASE 4.—R. G., aged 18, was admitted to the United States Naval Hospital, Brooklyn, N. Y., Sept. 30, 1943. He stated that during a routine physical examination in March, 1943,

a symptomless mass involving the left fourth rib was discovered. The tumor was removed at another Naval Hospital in May, 1943, and a diagnosis of chondroma was made. In September, 1943, he began suffering from episodes of hemoptysis and at this time he entered the Brooklyn Naval Hospital.

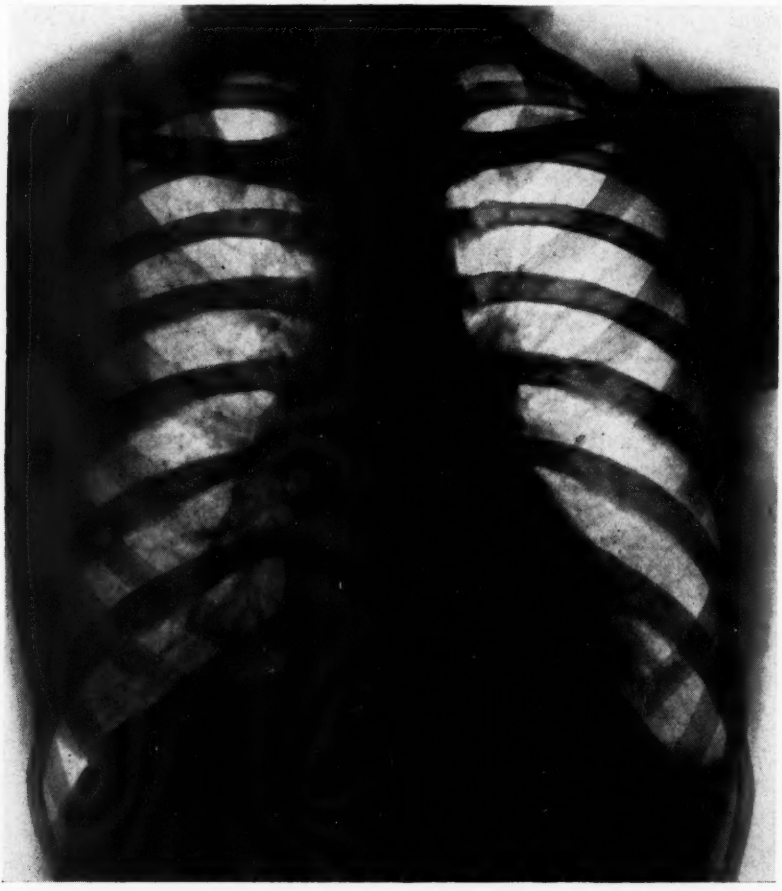

Fig. 5.—Roentgenogram eight days postoperatively.

Physical examination was essentially negative. There was a 4-inch, well-healed scar in the anterolateral aspect of the left chest in the area of the fourth interspace. There was no local evidence of recurrence. X-ray examination of the left thoracic cage showed evidence of a chondroma involving the partially-amputated anterior end of the fourth rib. After partial pneumothorax it was demonstrated that there was no fixation of the lung to the recurrent growth.

The patient was prepared for surgery and on Dec. 18, 1943, under cyclopropane, endotracheal anesthesia, a bony tumor of the left fourth rib together with the adjacent third rib was removed. This excision left a chest wall, pleural, and soft part defect which measured 8 x 10 cm. A fascia lata graft was next obtained and sutured into this defect.

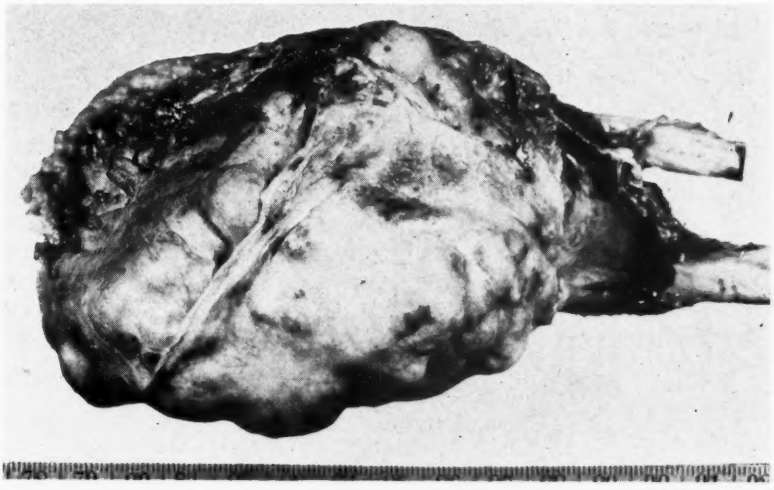

Fig. 6A.—Surgical specimen.

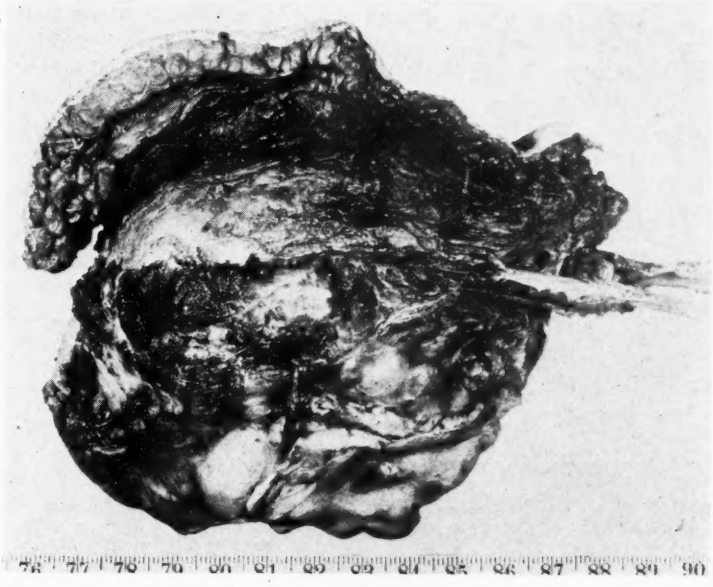

Fig. 6B.—Surgical specimen.

The postoperative course was essentially uneventful. There was some pleural reaction on the left side which subsided completely, and the incision healed per primum.

The final pathologic diagnosis was osteochondroma.

DISCUSSION

The use of fascia lata grafts in chest wall defects has several definite advantages:

1. It eliminates the need for a fixed, foreign body in the constantly expanding and contracting chest wall.

2. Fascia lata is readily available, even though it may not have been planned preoperatively to use a graft. If some other type of material were to be used, such as tantalum, definite preoperative plans would have to be made.

3. A better "cancer operation" can be carried out if one plans to help close the postoperative defect with a graft. A large graft of fascia lata will take as well as a small one, and one should not hesitate to resect at least one rib beyond the palpable extent of the tumor.

4. In cases of resection of the entire thickness of the chest wall, with exception of the skin, fascia lata adds a certain amount of support and also aids in making the chest wall airtight at time of operation.

5. Fascia lata grafts are firm enough to prevent postoperative pulmonary herniation.

The blood supply to the fascia lata grafts evidently comes through the side away from the pleural cavity. In Cases 2 and 3, the grafts took well in spite of varying amounts of pleural effusion. In the only case in which a postmortem examination was carried out, the fascia lata graft had taken well in spite of the presence of pleural fluid. Although, in these four cases, the procedure was used in the surgical care of primary tumors of the chest wall, it should also be applicable in cases of cancer of the breast with direct extension of disease into chest wall but without distant metastases.

SUMMARY

Four young patients had their large chest wall tumors resected and the resultant surgical defects closed with the aid of fascia lata grafts. The grafts took well in each case, and adequate chest wall support resulted in each instance. There was no evidence of postoperative herniation in any of the four cases.

REFERENCES

1. Hedblom, C. A.: Tumors of the Bony Chest Wall, Arch. Surg. **3**: 56-86, 1921.
2. Hedblom, C. A.: Tumors of the Bony Chest Wall, Ann. Surg. **98**: 528, 1933.
3. Sommers, G. N. J., and Major, R. C.: Neoplasms of the Bony Thoracic Wall, Ann. Surg. **115**: 51-83, 1942.
4. Effler, D. B., Captain, and Blades, B., Colonel: The Functions of Thoracic Centers in World War II, Am. J. Surg. **72**: 319-331, 1946.
5. Kirschner, M.: Present Status and Prospects of Autoplastic Transplantation of Fascia, Internat. Abstr. Surg. **18**: 371-372, 1914.
6. Brenner, E. C.: Reconstruction of a Portion of Male Urethra by Means of Fascia Lata Transplant, J. Urol. **11**: 197-198, 1924.
7. Gallie, W. E., and LeMesurier, A. B.: Use of Free Transplants of Fascia as Living Sutures in the Treatment of Hernia, Arch. Surg. **9**: 516-529, 1924.
8. Shearburn, E. W., and Mulford, E. H.: Penetrating Wounds of the Brain, Am. J. Surg. **72**: 441-448, 1946.
9. Zinninger, M. M.: Tumors of the Wall of the Thorax, Ann. Surg. **92**: 1043-1058, 1930.

PUNCTURE WOUNDS OF THE CHEST

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PATIENTS with chest wounds are usually cared for by surgeons, and most of the literature on this subject is surgical. Unfortunately, this literature gives the impression that most of the care of patients with puncture wounds of the chest is surgical. Actually, only a very small proportion of the patients receiving such wounds have, in our experience, required surgery, if simple suture of a sucking wound can be called a prerogative of any doctor, or, if need be, of any medical corpsman. These wounds stand in sharp contrast to those of the abdomen, for instance, which entail painstaking surgery.

This report concerns the treatment on the medical service of a naval advanced base hospital of 100 consecutive patients with puncture wounds of the chest uncomplicated by severe wounds in other parts of the body. The patients were admitted some days after they had been wounded (Table I). Very

TABLE I

	NUMBER OF DAYS ELAPSED BETWEEN DATE OF WOUND AND ADMISSION TO HOSPITAL																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	70
Number of patients (Total number of patients, 100)	0	1	8	12	12	12	6	7	5	6	6	8	4	2	2	3	0	3	1	1	1

commendable records, covering the period from wounding to admission, were received with the patients and pertinent data are drawn from them and from our own observations for use in the study. The patients were unselected, except that those with recognized empyema, a small proportion of the total, were usually sent directly to the surgical service.

In all cases, metallic bodies or fragments, or bone splinters, punctured four structures: the chest wall, one or more blood vessels, parietal and visceral pleuras, and lung. There was always some degree of pulmonary damage, usually hemorrhage into the pleural space, sometimes pneumothorax, and occasionally infection which subsequently led to empyema.

In forty of the patients a single bullet caused the wound. Thirty of these were accompanied by a wound of exit from the chest. In thirty-one the wound was caused by a single metal fragment, from shrapnel or bomb, and six of these had a wound of exit. In nine patients two or more fragments punctured the chest, but none of these produced wounds of exit. In twenty instances the cause of the wound was not known.

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Sucking wounds had been sutured before the patients arrived. Thirty-eight wounds were known to have been sutured, but how often suturing was required for physiologic and bacterial reasons and how often for cosmetic reasons is not known. Only two of the sutured wounds broke down sufficiently to allow communication with the chest cavity. Both appeared to result from empyema, rather than to cause empyema.

In all instances the period of shock had passed, and in nearly every instance the coughing of blood and of blood-stained sputum had ceased. A few patients were still distinctly dyspneic, but even in these, cyanosis was not conspicuous.

The physical findings varied widely, but usually included a healed wound of entrance, a healing wound of exit, the signs of fluid, and sometimes the signs of pneumothorax or consolidation. The roentgenologic examination of thirty-one of the chests showed the following: thirteen patients with free fluid only, the pulmonary collapse proportional to the amount of fluid; five patients with fluid and intrapulmonary metallic fragment; two patients with fluid and considerable air; one with fluid and atelectasis; one with fluid and consolidation; one with localized fluid only; one with "a slight pleural reaction"; one with a metallic fragment and a wedge-shaped area probably representing pulmonary hemorrhage; one with a single metallic fragment; and two with several metallic fragments, all without other signs; one with atelectasis only; and one entirely negative.

Roentgenologic examination of the chest was discontinued as a routine because x-ray usually failed to reveal important information not obtainable by physical examination. Its importance at a later stage is not denied.

Thoracentesis, other therapy, and the passage of time, usually lessened physical and roentgenologic findings.

Treatment was conservative. Good nursing care was stressed. Penicillin in doses of 20,000 units every four hours was usually continued for two to three weeks from the time of wounding, but this varied somewhat depending upon how "toxic" the patient was on admission. Sulfadiazine, either alone or in combination with penicillin, was used in some instances but for shorter periods than penicillin. Blood and plasma were restored as required. Oxygen was only occasionally necessary, though many of the men had required it in the first several days after being wounded (Table III).

Hemothorax and pneumothorax were reduced before admission in some cases but more commonly during the week after admission. At each aspiration of fluid one liter was withdrawn if it could be obtained and if distress by cough or pain did not interfere. Actually, the average successful (10 c.c. or above) aspiration yielded 622 c.c. If fluid was present but could not be aspirated, further trials of aspiration were delayed for one or more weeks and were then frequently successful. Some fibrin collections that at first prevented aspiration appeared later to autolyze. Sixty-seven of the 100 patients had one or more successful chest aspirations, counting those performed before the

TABLE II. HOSPITALIZATION AT BASE HOSPITAL

NO. DAYS	NO. PATIENTS
1- 5	0
6-10	27
11-15	17*
16-20	28
21-25	10
26-30	9*
31-35	1
36-40	1*
41-45	2*
46-50	1
51-55	2†
56-60	1†
61-65	1*
Total no. patients	100

*One of these patients had empyema.

†One of these patients was held in hospital for observation for a prolonged period, solely for study.

TABLE III. THERAPEUTIC PROCEDURES (OXYGEN, PENICILLIN, SULFATHIAZOLE) BETWEEN TIME OF WOUNDING AND DISCHARGE FROM THE ADVANCED BASE HOSPITAL

NO. DAYS OF THERAPY	OXYGEN (NO. PATIENTS)	PENICILLIN (NO. PATIENTS)	SULFATHIAZOLE (OR SULFADIAZINE) (NO. PATIENTS)
1- 2	6	0	13
3- 5	2	5	10
6-10	2	31	19
11-15	2	23	4
16-20	0	23	5
21-25	0	9	0
26-30	0	2	0
31-35	0	2	0
36-40	0	0	0
41-45	0	1	0
Therapy given but number of days unknown	12	0	0
No therapy given	56	2	42
Total data available	80	98	93
Data not available	20	2	7
Total number patients	100	100	100

TABLE IV. THERAPEUTIC PROCEDURES (THORACENTESIS, TRANSFUSION, PLASMA) BETWEEN TIME OF WOUNDING AND DISCHARGE FROM THE ADVANCED BASE HOSPITAL

NO. PROCEDURES PER PATIENT	THORACENTESIS (NO. PATIENTS)	BLOOD TRANSFUSION (NO. PATIENTS)	PLASMA UNIT (NO. PATIENTS)
1	23	24	16
2	18	11	20
3	11	10	2
4	6	4	2
5	4	5	1
6	2	1	0
7	0	1	0
8	3	0	0
None given	30	35	50
Data available	97	91	91
Data not available	3	9	9
Total no. patients	100	100	100

patients were admitted (Table IV). In one instance moderate secondary hemorrhage occurred after thoracentesis. In none did infection seem to result from thoracentesis.

Much of the blood and plasma used was administered before the patients were received, but some patients required one or both after admission (Table IV).

Metallic foreign bodies and bone splinters deep in the wounds were not removed. Those near the surface had been removed in some cases before the patients were admitted, and in some cases were removed afterward. Fibrin collections were removed surgically in only one instance (a patient with empyema) at the time of rib resection and initiation of open drainage. None of the lungs were "decorticated."

Fever of considerable degree was recorded in many patients who proved to have no empyema. We believe the fevers were usually the result of absorption of protein breakdown products of the hemothoraces, even though there was no correlation between the extent of the hemothorax and of the fever. In forty-five patients who developed no empyema the oral temperature was 101° F. or above, seven days or more after the patients were wounded. In twenty-five of these the peak of fever, seven days or more after the patients were wounded, was 102° to 102.8° F. In twelve others the peak was 103° to 105° F.

Sustained fevers above 103° F. strongly suggested the onset of empyema, and those patients required frequent diagnostic aspirations and culture of the fluid. When the fluid lost its thin appearance, contained shreds, had a foul odor, contained many pus cells in smear, and yielded bacteria on culture, there was no doubt that empyema had developed, and the patient was transferred to the surgical service for rib resection and drainage. The five patients in this category had peaks of fevers of 102.5°, 103.0°, 103.0°, 103.8° and 105.0° F. respectively, and all had empyemas. Four of the five empyemas yielded pure culture of *Staphylococcus albus* or *aureus*. When fever was moderate and the patient was not toxic, a single positive culture was not considered diagnostic of empyema because several patients with positive culture in the absence of confirmatory findings failed to develop empyema. Penicillin administered systemically appeared to have some bacteriostatic action in cases of suspected empyema that failed to materialize. On the other hand, once empyema was established, penicillin administered intramuscularly (20,000 units every fourth hour) and intrathoracically (100,000 units daily) had little effect. In one instance, intrathoracic penicillin appeared to lower the fever somewhat, but the daily drainage of fluid may have contributed to the effect.

Cardiac involvement was suspected clinically in seven cases. None of these patients required a pericardial tap and none evidenced pericardial sepsis. One of two who developed pericardial friction rubs had faint heart sounds and a cardiac silhouette by roentgen examination suggestive of fluid. Their electrocardiograms showed T-wave flattening or shallow inversion in various leads (Figs. 1 and 2).

Three patients, twelve to twenty-one days after being wounded, developed precordial or substernal pain with left shoulder radiation associated with dyspnea and signs of shock. Their tracings showed more marked abnormalities, consisting in two instances of biphasic T-waves in Lead I and sharply inverted T-waves in Lead IV-F (Figs. 3 and 4). The third patient had inverted T-waves in the limb leads with slight RS-T segment elevation in Leads II and III (Fig. 5, *A*). A tracing five days later showed deeper inversion in these latter leads with disappearance of the RS-T segment displacement (Fig. 5, *B*). We are unable to offer an explanation for the relatively late development of cardiac symptoms in these three cases.

The remaining two patients had no cardiac symptoms or clinical signs but the location of their wounds led us to suspect cardiac injury. The electrocardiograms of one showed T-wave inversions in Leads I, II and IV-F which became more marked after five days (Fig. 6, *A* and *B*). The other patient had a deep Q-wave in Lead I, smaller Q-waves in Leads II and IV-F and T-wave abnormalities most marked in Lead I (Fig. 7).

Electrocardiograms were taken on fifteen patients with no clinical evidence of cardiac or pericardial involvement. Nine had left, and six had right hemothoraces. Seven of the patients having left hemothoraces had normal tracings and two had transient diminution of T-wave amplitude. These latter had a mediastinal shift but so did three of the patients whose electrocardiograms were normal. Of the six patients with right hemothorax, three had right axis deviation and one had a vertical axis.

Thus electrocardiography consistently showed changes compatible with pericardial or myocardial abnormality in the 7 per cent of our patients having clinical signs or symptoms referable to the heart. Associated hemothorax and mediastinal shift did not produce misleading electrocardiographic changes.

The average duration of hospitalization of all of the hundred patients with chest wounds was nineteen days (see Table II). All but two of the patients appeared to improve. One of these two died. Most of the patients improved conspicuously. Four of those with empyema were free of fever when evacuated.

Evacuation was accomplished by air transport in most instances, but some was by hospital ship. Evacuation by ships not primarily designed for hospital work was avoided because patients with chest wounds might develop needs for surgery. Patients with pneumothorax were evacuated by hospital ship, and were not exposed to risk of expanding pneumothorax at increased altitudes.

The one patient who died was wounded fifteen days before admission to the advanced base hospital. A bullet had entered the third interspace at the left sternal margin and emerged from the left anterior axilla and had produced a sucking wound which had been sutured at an advanced field hospital. He denied hemoptysis. He was at first extremely dyspneic. One liter of bloody fluid was aspirated from the left chest. Eight days after he was wounded, x-ray examination showed almost complete collapse of the left lung, with a fluid level at the ninth rib posteriorly. At that time the temperature and pulse were only slightly elevated, and respirations were not severely difficult.

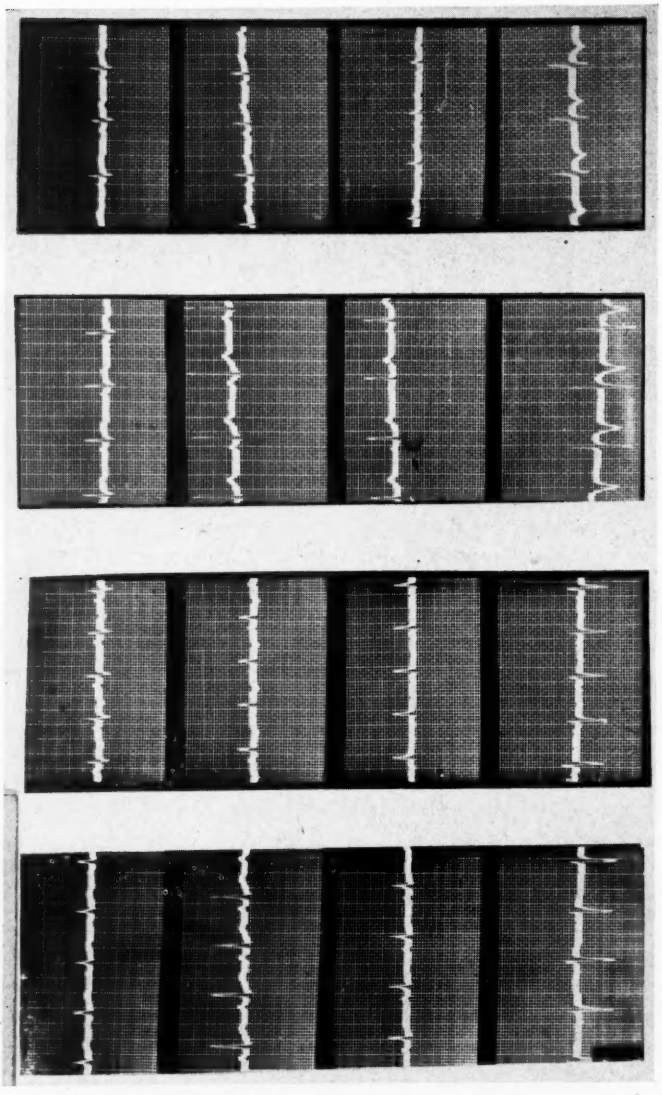

Fig. 1. Fig. 2. Fig. 3. Fig. 4.

Electrocardiograms taken on patients having cardiac injury as a complication of their chest wounds. For description, see text.

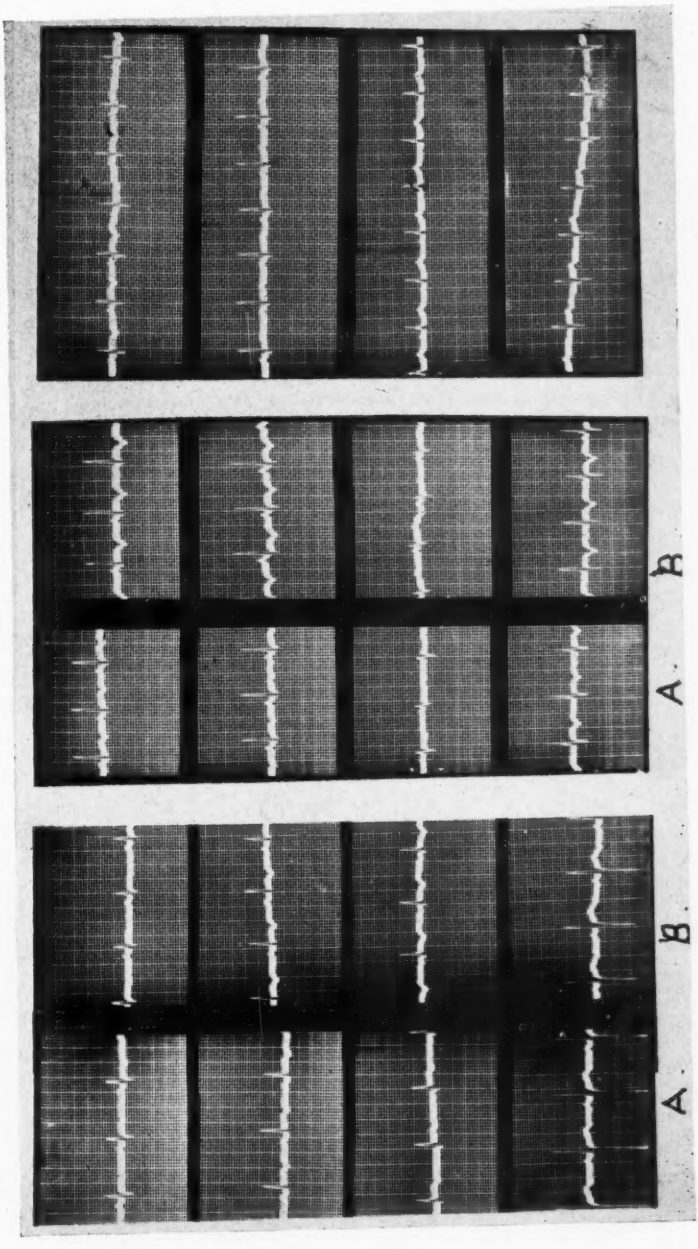

Fig. 5.

Fig. 6.

(For legend see opposite page)

Fig. 7.

During the following ten days, in spite of penicillin therapy, he had one, then two, and finally four and one-half degrees of fever. Only one cubic centimeter of a cloudy fluid was recovered by thoracentesis. He was transferred to the surgical service. Thoracotomy was performed, and drainage was established. The toxic state failed to diminish. Further exploration was unsuccessful. He died several days later. Autopsy showed a single, drained, left empyema cavity with no encapsulated pus, little or no expansion of the lung, some hemorrhage into the pleural cavity, and toxic hepatitis and splenitis.

DISCUSSION

Most patients with chest wounds resulting from this war have been handled in much the same way as were those in the present report.[1] Our cases are not presented because of originality. They are presented in order that one can understand what to expect of one phase of conservative therapy. Unfortunately, there is no opportunity at the present time to follow our patients through to complete recovery.

The treatment of chest wounds we have employed can best be called the conservative method. In all but the five instances in which the patients developed empyema, it was carried out on a medical service without benefit of surgery. In the case of the patients with empyema, usual rib resection and open drainage were performed by the surgeons.

Considering the shocking nature of the wounds, the patients improved very satisfactorily, and most were on their way to complete recovery when discharged from the hospital. In some instances in which fibrin collections prevented aspiration and interfered with re-expansion of the lung, the fibrin later autolyzed and expansion was accomplished by successful aspiration. In some instances fibrin collections did not autolyze. Empyemas were remarkably exceptional.

Other less conservative methods of treating puncture wounds of the chest have been used. Much has been written during the war years of surgical intervention to remove fibrin collections.[2 and 3] Its advocates suggest the third to tenth week as the optimum time for this, since some believe that irreversible encapsulation of incompletely expanded lung may result from fibrin collections within that time. Other surgeons, some of the British for example, believe major surgery should be utilized on the day of wounding, after the patient has recovered from shock.[4] They advocate exploration of the chest under closed anesthesia, suture of the bleeding vessels, evacuation of all blood and clot, rinsing of the pleural space with physiological saline, and maintenance of artificial pneumothorax. Most American surgeons insist that pneumothorax should be avoided, or reduced as promptly as possible.

Good results have been obtained by these surgical methods when carried out by competent thoracic surgeons. We doubt that they afford lowered mortality or shortened morbidity. Their practicality, especially at advanced bases in average hands, is not proved, and statistics of end results are incomplete.

Statistics concerning the relative validity of various methods of treating wounds of the chest could be obtained from the records of the medical corps of the services and from a follow-up of wounded to the time of recovery. This would help immeasurably in choosing the best methods of treating chest wounds.

SUMMARY

The conservative medical management of 100 patients with puncture wounds of the chest at an advanced naval base hospital has been outlined. All but two of the patients improved, and all but one lived. Five developed empyema and four responded well to surgical drainage. We hope this will stress the need for a statistical comparison of the end results of treatment by conservative methods and by surgical intervention.

REFERENCES

1. Ferguson, L. Kraeer, and Brown, Robert B.: Injuries of the Thorax in Battle Casualties, Am. Rev. Tuberc. 50: 293, 1944.
2. Notes on Care of Battle Casualties: Technical Bulletin Medicine 147, United States War Department, March, 1945. II. "Thoracic Wounds."
3. Blades, Brian, and Dugan, David J.: War Wounds of the Chest Observed at the Thoracic Surgery Center, Walter Reed General Hospital, J. THORACIC SURG. 13: 294, 1944.
4. Davies, H. Morriston and Coope, Robert: War Injuries of the Chest, Baltimore, 1942, Williams and Wilkins Company.

SODIUM TETRADECYL SULFATE, AZOCHLORAMIDE, AND SULFANILAMIDE MIXTURE IN THE PREVENTION OF EMPYEMA FOLLOWING PNEUMONECTOMY WITH CONTAMINATION

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ENCOURAGING results in attempts to sterilize the pleural cavity in pyogenic empyema with instillations of an antiseptic solution[3, 10] led the authors to extend its use to the prevention of empyema following pneumonectomy.

Contamination of the pleural cavity during pneumonectomy in cases of lung abscess, bronchiectasis, or carcinoma complicated by lung abscess is a great hazard in thoracic surgery. The empyema which usually follows such contamination presents a serious problem in treatment. Patients afflicted with such a condition continue to drain for months, or death from sepsis and debilitation may occur. Following prolonged drainage of the pleural cavity, obliteration by thoracoplasty offers the best chance for recovery. Complete obliteration can only be accomplished by the multiple stages of a thoracoplasty. This is a costly, time-consuming, and sometimes hazardous procedure, especially for patients who already have been ill, perhaps for many months, and who have been subjected to pneumonectomy.

The search for a solvent for digestion of sputum and empyema fluid in the concentration examination for tubercle bacilli[5, 6] led to the use of detergents or wetting agents. Of the numerous detergents used in experiments, sodium tetradecyl sulfate was the most satisfactory. It had been known for many years that soaps enhanced the effect of bactericidal agents. The wetting agents which possess soap properties in a greatly intensified degree were then selected as a basis for further study in combination with antibacterial solutions.

The properties of the wetting agents explain their effectiveness for this purpose. Because of the dual nature of the structure of their molecules, wetting agents have a tendency to locate themselves on an aqueous surface or interface, thus forming a monomolecular film. For this reason, the surface tension of solutions coming in contact with the agent is reduced. Highly surface active solutions can, therefore, penetrate into openings and cracks, crypts and recesses, which a conventional aqueous saline solution would simply bridge. Solutions of wetting agents can even wedge their way into the center of clumps of bacteria, leucocytes, and debris.

With these ideas in mind Petroff and Schain[4] experimented with the bactericidal properties of various antiseptics reinforced with different detergents. Of the many combinations of compounds used, azochloramide and sodium tetradecyl sulfate solution were found to be the most effective. Animal experiments

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as well as clinical observations have shown that sodium tetradecyl sulfate is well tolerated by mammals when administered orally, intravenously, or when injected into the pleural cavity.[1, 2]

Following the experimental work of Petroff and Schain, Palitz and Herman[7] described the treatment of tuberculous and mixed infection empyema with azochloramide-sodium tetradecyl sulfate solution. Successful results were noted in 81.5 per cent of the cases without bronchopleural fistulas. In general, following the irrigations they reported that the fever was lowered and the fluid was thinned. Sinuses and abscesses were affected favorably.

Brantigan and Owings[3] followed up this work and reported the use of sodium tetradecyl sulfate and azochloramide in the treatment of acute pyogenic empyema of the pleural cavity. Two patients were treated and the results proved outstanding.

In 1941, Neter[8] reported on the combined bacteriostatic activity of sulfanilamide and azochloramide upon Group A hemolytic streptococcus and enterococcus. His experiments revealed that sulfanilamide (1,000 mg. per cent) used in combination with azochloramide (1:800,000) may prevent completely the growth of enterococcus and exert greater bacteriostatic activity than azochloramide alone, even in a concentration twice as great.

Schmelkes and Wyss[9] corroborated this work. Their experiments indicated that the bactericidal effect of sulfanilamide in the test tube can be enhanced greatly by small concentrations of azochloramide which are ineffective in themselves. Similar concentrations of azochloramide will also restore the bactericidal effect of sulfanilamide in the presence of para-aminobenzoic acid (which neutralizes the action of sulfanilamide), suggesting that the enhancement observed in the first place is attributable to the inactivation of sulfanilamide inhibitors by the azochloramide.

The results of the work of these men indicate that the combination of sodium tetradecyl sulfate, azochloramide, and sulfanilamide is a highly effective nonspecific bactericidal agent. The authors have placed it in the pleural cavity at the termination of all open chest operations since 1943. In uncontaminated thoracic operations it seems to have eliminated all empyema. However, there is no proof that infection would have occurred without its use.[1, 2] The mixture seems also to reduce pleural exudation which, of course, reduces or eliminates the necessity for postoperative pleural aspirations following open thoracic operations.

Since adopting the routine use of the mixture in the pleural cavity after open chest operations, four cases of gross contamination of the pleural cavity occurred during pneumonectomy. The results in these four cases were so highly satisfactory that they are being reported.

CASE REPORTS

CASE 1.—W. S. was a white man aged 32. In 1942 a carcinoma of the testicle was removed. A submucous nasal resection was performed in March, 1944. In October of the same year a diagnosis of disease in the right lung was made; malignant metastasis from the testicle was suspected. The disease in the right lung became more extensive, and the patient, weaker. The left lung was found to be normal. Based upon the possibility that malignancy

might not be the cause of his condition, operation was advised and accepted. By this time the presence of multiple lung abscess cavities was definite. On May 2, 1945, under general endotracheal anesthesia, a right pneumonectomy was performed. During operation one lung abscess cavity was entered and foul-smelling pus was spilled into the pleural cavity. Sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 in 200 c.c. quantity and 5 Gm. of sulfanilamide were placed in the pleural cavity. The chest was closed without drainage. The patient's course was uneventful and his temperature normal until the third postoperative day, when he began to have slight fever. Empyema of the right pleural cavity could not be diagnosed until the eighth postoperative day.

After the diagnosis of empyema, aspirations and instillation of the sodium tetradecyl sulfate, azochloramide and sulfanilamide mixture would eliminate his symptoms but would not cure the infection. A pure culture of *Staphylococcus aureus hemolyticus* persisted. Open drainage of the pleural cavity was done, followed by a multiple stage thoracoplasty approximately six months later. A satisfactory cure has resulted. There was no sign of malignancy in the lung that was removed.

Comment.—From a study of the patient it appeared evident that the single application of the antiseptic solution killed all bacteria except the *Staph. aureus hemolyticus,* inhibited the growth of these bacteria for three days, and definitely retarded their growth for several more days. The solution failed to kill all the bacteria and true empyema developed. It seemed logical to believe that perhaps too little solution had been used. The antiseptic, of course, was diluted by blood and serum oozing into the pleural cavity and perhaps even changed in chemical consistency. It was decided that when the next gross contamination of the pleural cavity occurred with pneumonectomy a larger amount of the solution would be used, followed by aspiration of the pleural cavity and instillation of the sodium tetradecyl sulfate, azochloramide and sulfanilamide mixture each day after operation until three such treatments had been given.

CASE 2.—J. C. was a colored man, aged 43. In June, 1945, his chief complaint was pain in the left side of the chest accompanied by fever for a few days. A roentgenogram made at this time revealed a lesion in the region of the hilus of the left lung, with ill-defined cavities. Sputum studies failed to show tubercle bacilli. Bronchoscopic examination then confirmed the impression of multiple lung abscesses. On admission to the hospital the patient was running a febrile course with daily elevations of temperature to 102° F. by mouth. Operation for pneumonectomy was advised and accepted by the patient. Therefore, on Nov. 11, 1945, under general endotracheal anesthesia, a left pneumonectomy was performed. During the operation the major bronchus of the left lung was accidentally opened, thus spilling foul-smelling pus into the pleural cavity. Prior to the closure of the chest without drainage approximately 500 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 with 5 Gm. of sulfanilamide was placed in the pleural cavity. On the first, second, and third postoperative days the chest was aspirated and instilled with the same mixture. The patient's postoperative course was uneventful. His temperature was normal on the seventh postoperative day. The wound did not drain and the pleural cavity did not become infected. He was discharged Dec. 2, 1945, and has remained well. The pathologic diagnosis was multiple abscesses of the left lung.

CASE 3.—S. P. was a 46-year-old white man who was admitted to the hospital with diagnosis of abscess in the upper lobe of the left lung. The patient reported that his present illness began in December, 1945. Following admission to the hospital he ran a low-grade fever for three weeks. On his twenty-fifth hospital day he experienced an episode of sharp pain in the left side of the chest accompanied by increased temperature elevation, accelerated pulse rate, and bloody sputum. A physical examination showed evidence of thrombophlebitis of the left femoral vein. Therefore, on Aug. 15, 1946, a ligation of the common iliac vein

on the left side was performed. It was postulated at this time that the patient's lung abscess may have been caused by a previous pulmonary infarct. It was decided to watch the patient further in the hope that his lesion might spontaneously improve following obliteration of the source of the emboli. He was observed for a two-month period, during which time he periodically ran a low-grade fever and the chest lesion seemed to increase in size.

Exploratory thoracotomy was advised and accepted by the patient. On Oct. 8, 1946, under general endotracheal anesthesia, the left pleural cavity was explored. An examination of the upper lobe of the left lung revealed a lesion that was thought to be a bronchogenic carcinoma complicated by a lung abscess. A left pneumonectomy was performed; however, during the course of the operation the left pleural cavity was grossly contaminated by several cubic centimeters of foul-smelling pus which was spilled when the abscess cavity in the lung was entered inadvertently. Prior to closing the chest without drainage 500 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1,3,300 with 5 Gm. of sulfanilamide was instilled into the pleural cavity.

The patient's temperature, which had been 101° F. several days prior to operation, was 100° F. on his first postoperative day. The chest was aspirated at this time and 400 c.c. of bloody fluid removed; 300 c.c. of sodium tetradecyl sulfate, azochloramide, and sulfanilamide mixture were instilled into the left pleural cavity. On the second postoperative day his temperature reached 102° F. and the same procedure was repeated. On the third postoperative day his temperature rose to 101° F. Thoracentesis was repeated and 350 c.c. of bloody fluid was aspirated; 300 c.c. of sodium tetradecyl sulfate, azochloramide and sulfanilamide was placed in the pleural cavity. On the fourth postoperative day the patient's temperature returned to normal, where it remained for the duration of his stay in the hospital. The wound did not become infected, nor did infection of the pleural cavity occur. The patient was discharged on his twenty-fifth postoperative day. The pathologic diagnosis was bronchiogenic carcinoma with bronchiectasis and abscess formation.

CASE 4.—J. M. was a 35-year-old white man who was admitted to the hospital with a diagnosis of bronchiectasis of the left lung. His past history was significant because in 1918 he had bilateral pneumonia followed by empyema of the left pleural cavity. An operation for open drainage was performed; the patient recovered and was asymptomatic until 1932, when he had hemoptysis. He continued to cough blood occasionally and in the last few years prior to admission hemoptysis had become more frequent. Bronchography revealed an extensive cystic type of bronchiectasis throughout the left lung from the apex to the base. The right lung was normal. The heart was markedly retracted to the left side.

On Oct. 18, 1946, under endotracheal anesthesia, a pneumonectomy of the left lung was performed. During the operation, which was accomplished with great difficulty, it was evident from the appearance in the chest that a bronchiectatic abscess cavity had been entered. This was confirmed by examination of the lung after completion of the operation. Prior to closure of the chest without drainage 1,000 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 with 5 Gm. of sulfanilamide was placed in the pleural cavity. On the following day 700 c.c. of bloody fluid were aspirated and 200 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 with sulfanilamide were inserted into the pleural cavity. On the second postoperative day 450 c.c. of bloody fluid were aspirated and 200 c.c. of the sodium tetradecyl sulfate-azochloramide solution with sulfanilamide were instilled. On the third day the same treatment was repeated. During this period the patient ran a low-grade febrile course with occasional elevations of temperature to 101° F. by mouth. On the fifteenth postoperative day 450 c.c. of serosanguineous fluid were again aspirated. No instillation was done. The patient remained free of wound infection or empyema and was discharged from the hospital on the twenty-fifth postoperative day. The pathologic diagnosis was bronchiectasis of the left lung.

Comment.—All the patients, except in Case 1, received 30,000 units of penicillin by intramuscular injection every three hours. However, penicillin

was not placed in the pleural cavity at the time of operation, nor was it instilled later into the pleural cavity in any of the four cases reported.

One important aspect is obviously deficient in this report. Cultures of the pleural cavity were not obtained at the time of operation. Nevertheless, in each case several cubic centimeters of pus were spilled into the pleural cavity. Subsequent cultures of the aspirated pleural fluid were absent when the charts were reviewed. The few culture reports available have been omitted purposely because they are incomplete and have a tendency to strengthen the beneficial effects noted clinically.

This report seems justified since the method of treatment has succeeded in preventing empyema in three consecutive patients where gross contamination of the pleural cavity occurred during pneumonectomy. These patients have been followed since their discharge from the hospital and all have remained well.

In Case 1 the solution would not cure an established empyema caused by hemolytic *Staph. aureus*. However, it is important to point out that a pleural space that cannot be obliterated, as one finds after pneumonectomy, presents an entirely different problem from empyema where there is a lung that can expand enough to finally obliterate the pleural space. Where a lung is present to obliterate the pleural space, even though a few bacteria survive the antiseptic they will be trapped and killed off by the body defenses. In an endeavor to sterilize chronic empyema cavities where obliteration of the pleural space could not be accomplished, the solution usually will quickly eliminate all bacteria except hemolytic *Staph. aureus* or *albus*. It will keep these inactive so long as the solution is changed at intervals of forty-eight hours.

In the three cases presented the treatment is directed at killing the contaminating bacteria before infection or empyema can become established. It seems logical that an attack upon any bacteria at this stage would be more likely to eliminate them than an attack made after they had successfully invaded the tissues. Of course, it is possible that hemolytic *Staph. aureus* or *albus* was not present in the pus contaminating the pleural cavity of the cases reported.

Since sulfanilamide is poorly soluble, it is impossible to determine how much of the drug enters the pleural cavity upon the instillation with syringe and needle. Usually 5 Gm. of sulfanilamide was placed in 500 c.c. of the solution of sodium tetradecyl sulfate 1:500 and azochloramids 1:3,300. The mixture is agitated and then drawn into the syringe and instilled; thus, only an indefinite amount is actually deposited in the pleural cavity. However, with the chest open at the operating table, the sulfanilamide is readily deposited as desired. The mixture should be prepared fresh prior to usage, since azochloramide is an unstable preparation. In aqueous solution this compound decomposes and gives up its chlorine; it therefore decreases in potency when left exposed to air and light. The mixture should be used at approximately body temperature. The sudden introduction of this solution into the pleural cavity may bring about temporary respiratory arrest in the anesthetized patient. The period of apnea is of short duration, but it is well for the anesthetist to realize that a period of apnea may occur.

CONCLUSIONS

Sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,000 with sulfanilamide is an efficient nonspecific bactericidal agent.

When the mixture was properly applied to the pleural cavity after pneumonectomy, it prevented empyema in three consecutive cases where gross contamination occurred during pneumonectomy.

REFERENCES

1. Brantigan, O. C.: Resection of the Lung in the Treatment of Pulmonary Tuberculosis, Arch. Surg. **51**: 147-153, 1945.
2. Brantigan, O. C.: Open Pneumonolysis, J. THORACIC SURG. **15**: 341-348, 1946.
3. Brantigan, O. C., and Owings, J. C.: Sodium Tetradecyl Used in the Treatment of Acute Pyogenic Empyema of the Pleural Cavity, Bull. School of Medicine, Univ. of Md. **26**: 247-253, 1942.
4. Petroff, S. A., and Schain, Philip: The Enhancement of Bactericidal Properties of Well Known Antiseptics by Addition of Detergents, Quart. Bull. Sea View Hosp. **5**: 372-384, 1940.
5. Petroff, S. A., and Schain, P.: New Concentration and Staining Method for Demonstrating Acid-Fast Bacilli in Sputa and Other Tuberculous Matter, Quart. Bull. Sea View Hosp. **4**: 145, 1939.
6. Petroff, S. A., and Schain, P.: Sputum Examination; Additional Data Pertaining to Digestion and Staining, Quart. Bull. Sea View Hosp. **5**: 183, 1940.
7. Palitz, L., and Herman, M.: Treatment of Tuberculous and Mixed Infection Empyema With New Irrigating Solutions, Quart. Bull. Sea View Hosp. **6**: 181-196, 1941.
8. Neter, E.: Combined Bacteriostatic Activity of Sulfanilamide and Azochloramide upon Group A Hemolytic Streptococcus and Enterococcus, Proc. Soc. Exper. Biol. & Med. **47**: 303-305, 1941.
9. Schmelkes, F. C., and Wyss, O.: Inactivation of Sulfonamide Inhibitor by Azochloramide. From the Research Laboratories of Wallace and Tiernan Products, Belleville, N. J.
10. Tonolla, E. H., and Brantigan, O. C.: Treatment of Thoracic Empyema With Sodium Tetradecyl Sulfate, 1:500 in Azochloramide, 1:3300, J. Dis. Chest. **13**: 123, 1947.

PULMONARY TUBERCULOSIS ASSOCIATED WITH ANOMALOUS COMMON LEFT PULMONARY VEIN ENTERING THE LEFT INNOMINATE VEIN

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THE perfection of technique for resection of the lung and its segments has called particular attention to the structural relationship about the roots of the lungs. This increased interest has been responsible for the introduction into the surgical literature in recent years, of various anomalies of the pulmonary circulation which have been encountered at operation and in the autopsy room.

Zuckerkandl,[1] in 1881, called attention to normal communications between the systemic and pulmonary circuits, and Brown[2] developed the present embryologic conceptions that explain the presence of wide variations from the normal in the termination of the pulmonary veins. Harris and Lewis[3] mention Brown's study of the development of the two vein systems from an undifferentiated celomic plexus, and state that in the adult, anomalies of the pulmonary veins will be of two types: in the upper chest there will be communications between the pulmonary veins and tributaries of the superior vena cava; and in the lower chest there will be those between the pulmonary veins and the hepatic portion of the inferior vena cava. The persistence of these embryologic communications is due, they believe, to inadequate development of the proximal portion of the cephalic tap (which later becomes the common pulmonary vein) resulting in the maintenance of the distal communications with the anterior or posterior cardinal vein or the caudal tap, the latter the precursor of the portal vein.

Maude Abbott[4] mentions the occurrence of anomalous pulmonary veins associated with cardiac defects and cites one case with a left pulmonary vein entering the left subclavian and the right pulmonary vein entering the portal vein. She states that wide variation from the normal may be associated with few symptoms. Brody[5] was able to collect 100 cases from the literature and added two of his own in which abnormal pulmonary veins were recorded. Many of them were associated with heart defects. He stated that, if 50 per cent of the pulmonary blood reached the left side of the heart, an adequate oxygen level would be sustained. Of fifteen such cases, he notes one, which was reported by Dean and Fox,[6] of a person who lived 86 years. This man had a common left pulmonary vein entering the left innominate vein. Compere and Forsyth[7] believe that if 75 per cent of the pulmonary blood reaches the left side of the

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heart, there will be no recognizable cardiorespiratory symptoms. Their case of an anomalous right upper lobe vein which entered the superior vena cava showed no circulatory difficulty during a fifteen-year period of observation for central nervous system syphilis.

This report concerns an anomalous common left pulmonary vein entering the left innominate in a patient with pulmonary tuberculosis under observation for over three years.

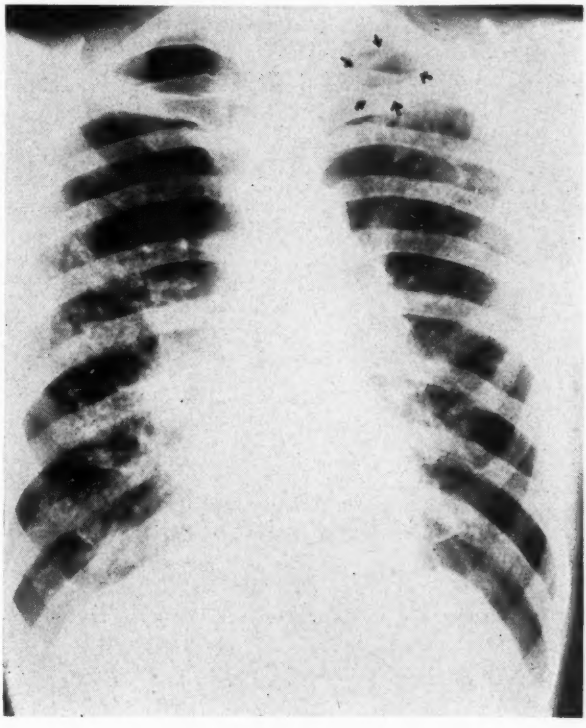

Fig. 1.

CASE REPORT

J. C., aged 37, was a Negro male, unskilled laborer. Bilateral pulmonary tuberculosis was discovered Nov. 27, 1942, when the patient was examined by x-ray prior to a hemorrhoidectomy at Freedmens' Hospital, Washington, D. C. (Fig. 1). The x-ray film showed fine nodular infiltration extending outward from the root into the middle third of the right lung field. The left lung showed scattered nodular infiltration above the third rib, anteriorly, with a 3 cm. cavity at the apex.

The lesion on the right side gradually improved but the left apical cavity increased in size until it extended down to the second rib, anteriorly. Left pneumothorax was induced in May, 1944. This was complicated by extensive, inoperable, apical adhesions and the pneumothorax was abandoned. In January, 1945, the left apical cavity had increased to 5 x 10 in size, and the lesion in the right lung had nearly completely resolved. In January,

1946, the left apical cavity occupied the entire apical region above the third rib, and the left lung began to show reactivation between the second and fifth ribs. During this period the man was a patient on the tuberculosis service of Freedmens' Hospital, but in March, 1946, it was necessary to discharge him as a disciplinary problem. He worked as a boot-black for about a month and then sought hospitalization when acute symptoms developed. In reviewing the case, Freedmens' Hospital noted no respiratory embarrassment during the entire stay at that institution.

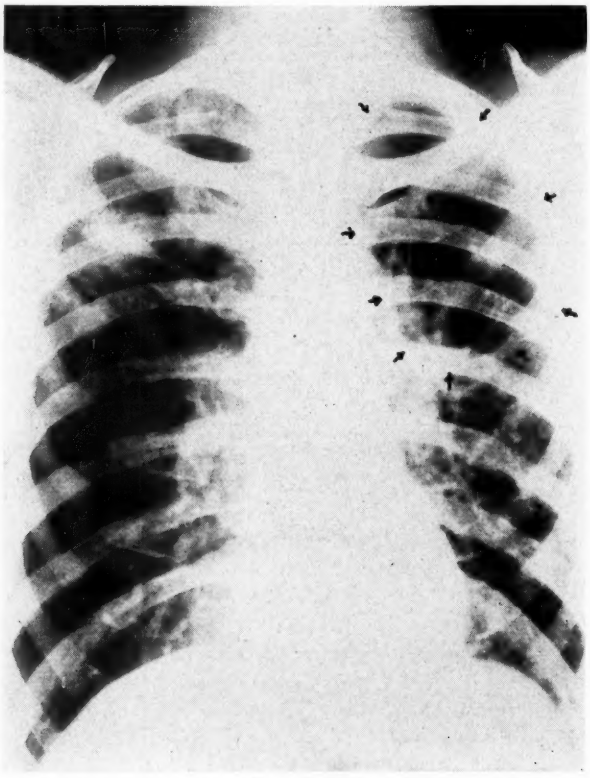

Fig. 2.

On admission to Glenn Dale Sanatorium May 3, 1946, the patient was critically ill, febrile, toxic, and dyspneic. The blood pressure was 136/88. There was no clubbing of the fingers and no evidence of cardiac disease (Fig. 2). The x-ray showed soft, mottled infiltration above the third rib, anteriorly, on the right, with the presence of several small cavities. The left lung contained a giant cavity above the third rib, anteriorly, with fine scattered infiltration to the base. His condition did not improve on bed rest, he developed a right pleural effusion, and died July 17, 1946.

Autopsy was performed July 20, 1946. The right lung was found densely adherent to the chest wall except at the base, where there was a pocket of clear pleural fluid about one liter in amount. Injection and tubercle formation were present over both visceral and parietal pleurae in this region. On section the lung showed marked consolidation throughout with several small cavities in the upper lobe.

The left lung was densely adherent to the chest wall and showed a 10 cm. apical cavity in the upper lobe and much caseation throughout the remainder of the lung parenchyma.

The heart was slightly enlarged, showing moderate dilatation of the right side and scattered small areas of atheromatous change in the aorta. There were no defects in the interauricular or interventricular septa. The left auricle received only one pulmonary vein, that from the right side, which divided as it passed through the pericardium. The circulation of the right lung was normal. Further exploration of the pulmonary circulation showed a common left pulmonary vein emptying into the left innominate and picking up a tribu-

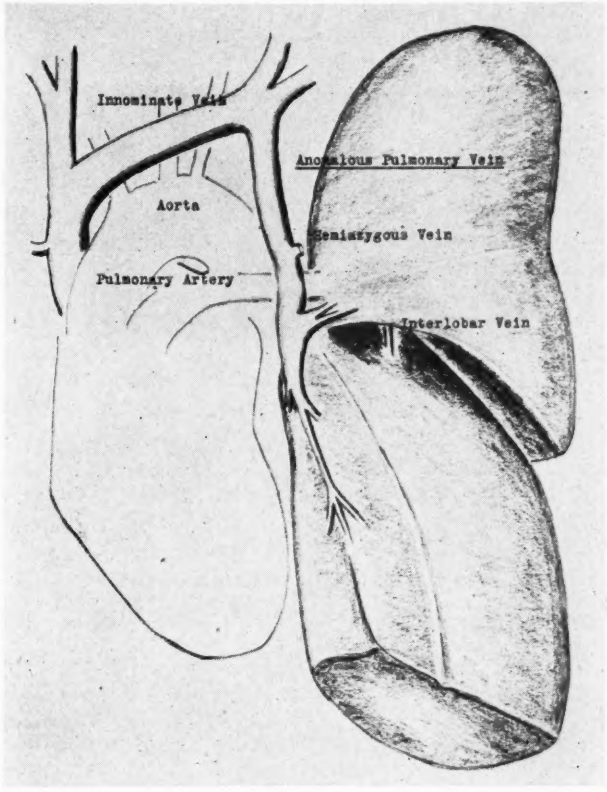

Fig. 3.

tary from what appeared to be the left hemiazygous above the level of the left pulmonary artery (Fig. 3). Except for its origin in the interlobular fissure in the cardiac sulcus of the lower lobe, the anomalous vein was entirely superficial, running anterior to the pulmonary artery, then proceeding along the mediastinum to its termination. The vein to the upper lobe was exceedingly short. One interlobar vein was found.

Extrapleural dissection was required to expose the left lung intact, and it is possible that communications with other veins of the chest wall may have been destroyed in the process.

COMMENT

It would appear that by itself recirculation of aerated blood from one lung into another, by a shunt involving half the pulmonary bed, still provides an

adequate pulmonary reserve. In the case reported, the arterial blood received all its oxygen from the right lung. The addition of pulmonary disease to the picture suggests in this instance that the integrity of the right lung was more important than the left. Obstruction to the flow of blood through the right lung, which was directly concerned with maintaining the oxygenation of the systemic circuit, had a more serious effect than involvement of the left lung. Up to the time the patient suffered his terminal episode, the tuberculosis was more extensive on the left side. When spread to the right lung occurred and was associated with a right-sided effusion, the supply of oxygen and blood to the systemic circuit then became limited.

The surgical implications of this anomaly are apparent. Because of the density of the mediastinal adherence about the left root, superficially, this anomaly had the appearance of the left superior vena cava reported by Lam.[8]

REFERENCES

1. Zuckerkandl, E.: Quoted by Brown.
2. Brown, A. J.: The Development of the Pulmonary Veins in the Domestic Cat, Anat. Rec. **7**: 299, 1913.
3. Harris, H. A., and Lewis, I.: Anomalies of the Lungs With Special Reference to the Danger of Abnormal Vessels in Lobectomy, J. THORACIC SURG. **9**: 666, 1940.
4. Abbott, Maude E.: Congenital Cardiac Disease, Osler's Modern Medicine, Philadelphia, 1927, Lea & Febiger.
5. Brody, H.: Arch. Path. **33**: 221, 1942.
6. Dean, J. C., and Fox, G. W.: Left Pulmonary Vein Emptying into Left Innominate, Wisconsin M. J. **27**: 120, 1928.
7. Compere, D. E., and Forsyth, H. F.: Anomalous Pulmonary Veins, J. THORACIC SURG. **13**: 63, 1944.
8. Lam, C. R.: Large Anomalous Vein ("Left Vena Cava") Encountered at Operation for Ligation of Patent Ductus Arteriosus, J. THORACIC SURG. **14**: 393, 1945.

ANOMALY OF AORTA SIMULATING MEDIASTINAL TUMOR

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SOLITARY tumors of the mediastinum tax the diagnostic acumen of the clinical surgeon. Symptoms referable to the tumor may be either absent or of little importance. A careful history and physical examination usually fails to disclose the presence of a tumor in the mediastinum.

Roentgenologic examination is our most important method of diagnosis. It frequently directs attention to unsuspected mediastinal pathology. In addition, it offers valuable information pertaining to the type of tumor by localizing the mass, by revealing its relationship to adjacent structures, by depicting its configuration, and by establishing the presence or absence of pulsation. Diagnostic roentgen therapy also plays a role in the differential diagnosis in certain instances, by determining radiosensitivity.

All too frequently the preoperative studies fail to establish with any degree of accuracy the type of tumor present. In a group of 114 patients operated upon for mediastinal tumors at a number of Army Thoracic Surgery Centers during the last war,[1] five were found to have unsuspected aneurysms, and in five cases benign inflammatory lesions were encountered. One of us, F. P. C., in an unpublished series of nineteen mediastinal tumors which were operated upon, was able to determine the cellular nature of the pathology preoperatively in eleven cases. In seven cases the preoperative prediction of the cell type was erroneous, and an aneurysm was encountered in one patient. We are reporting a case of unusual interest in which a unique anomaly of the aorta was found which simulated a mediastinal tumor.

CASE REPORT

The patient, a 58-year-old male, was admitted to the Veterans' Administration Hospital, Richmond, Va., on April 1, 1946, with a history of frequent "colds," weakness, and occasional pains in the chest. There had been some slight weight loss. He had been told that he had heart disease and had been advised to limit his activities.

Physical examination revealed a man of small stature. There was generalized arteriosclerosis of the medium-sized vessels. No cardiac enlargement was present. The blood pressure was 172 mm. of mercury systolic and 110 mm. of mercury diastolic. Fundoscopy revealed some blurring of the disk margins and some accentuated vascular markings on the temporal side of the disk on the left. Right fundoscopy was normal. The electrocardiogram demonstrated left axis deviation. Routine laboratory work-up was negative.

Roentgenographic examination of the chest revealed normal lung fields, no enlargement of the heart, and a dense mass in the posterior mediastinum extending from the level of the seventh rib to the tenth rib. Its convex border bulged into the left hemithorax. An overexposed posterior-anterior view of the chest emphasized the density of the "tumor mass"

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(Fig. 1). A lateral view of the chest showed an ill-defined tumor with its convexity posterior (Fig. 2, *A*). Fluoroscopic examination and a roentgenogram of the barium-filled esophagus showed the distal one-third of this organ displaced anteriorly and to the left (Fig. 2, *B*). There was no erosion of the vertebral bodies. There was some question of pulsation of the

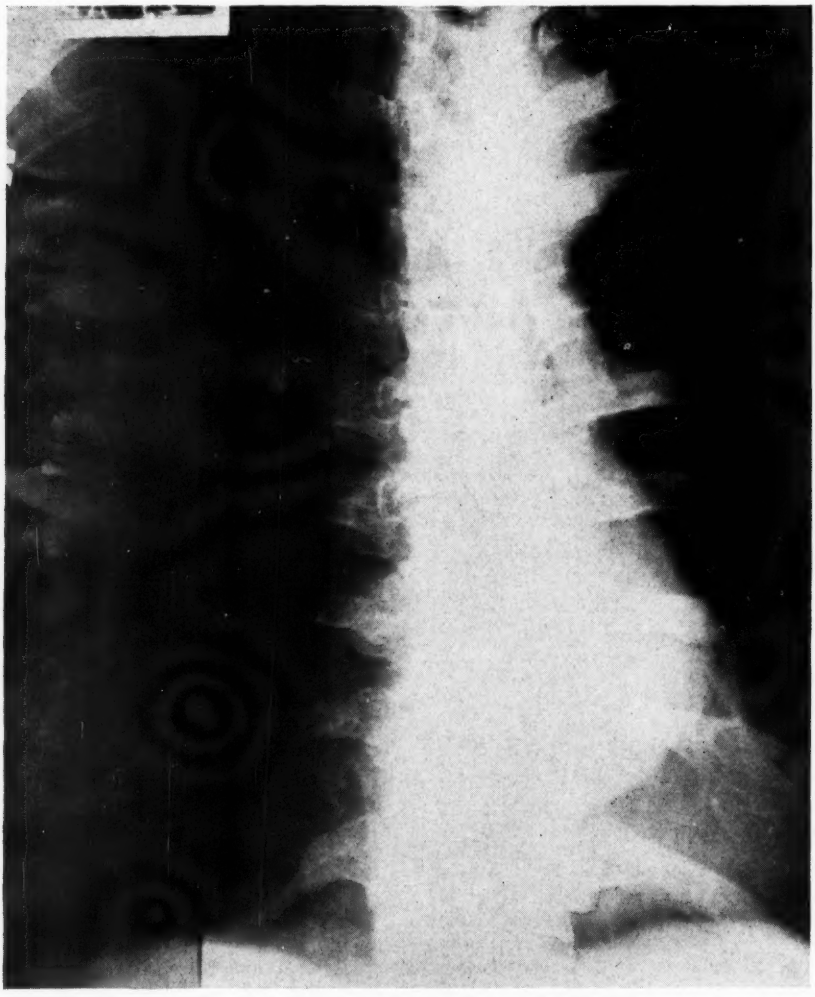

Fig. 1.—Overexposed posterior-anterior view of chest showing "tumor mass" extending from the level of the seventh rib to the tenth rib posteriorly.

mass during fluoroscopy, but kymographic studies were not available to confirm this observation. A study of the upper gastrointestinal tract with barium revealed a slight filling defect of the duodenal bulb. The cholecystogram was negative.

The differential diagnosis was limited to a consideration of an aortic aneurysm and a soft-tissue mediastinal tumor. The anatomic location and the questionable pulsation favored

the former, although sacular aneurysms of this portion of the aorta are seen infrequently. The absence of erosion of the vertebral bodies and the presence of an ill-defined mass with a posterior convexity extending into the costovertebral gutter (Fig. 2, *A*) made it necessary to consider mediastinal tumor.

On May 6, 1946, under endotracheal ether anesthesia, the left pleural cavity was opened through the periosteal bed of the resected eighth rib. The "tumor mass" was encountered adjacent to the angle of the resected rib. Further exploration revealed the mass to be an anomalous aorta. At the level of the seventh thoracic vertebra the arota deflected sharply to the left and took a horseshoe course before returning to the midline to pass through the aortic hiatus in the diaphragm (Fig. 3). The mediastinal pleura was opened in order to visualize the aorta to better advantage. There was marked arteriosclerosis of this vessel, but there was no increase in its circumference. The tortuosity which was most evident in the lateral plane was also present in the anterior-posterior diameter, thus accounting for the anterior displacement of the esophagus (Fig. 2, *B*). The left lung was normal.

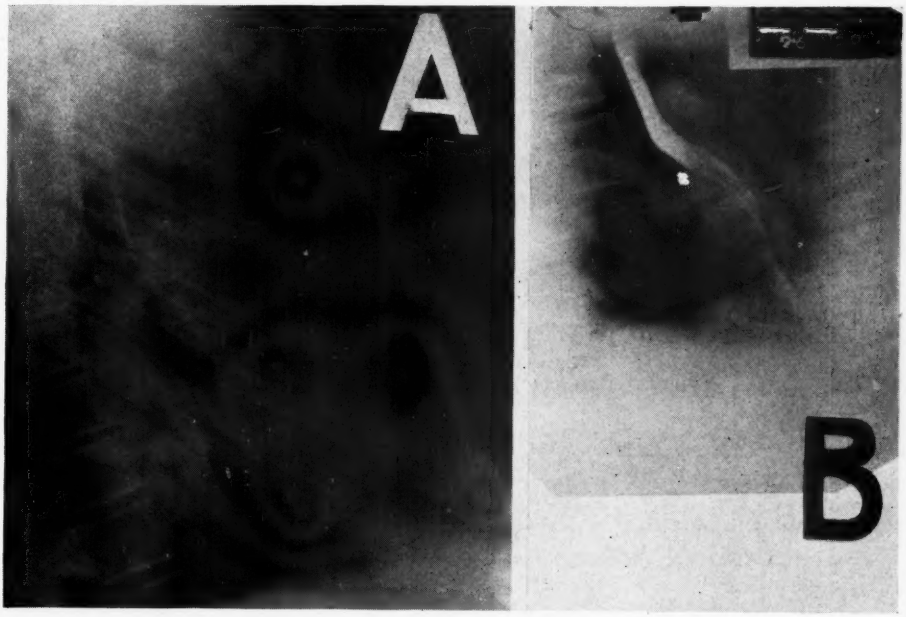

Fig. 2.—*A*, Left lateral view of chest "mass" ill defined. Note posterior convexity of tumor mass outlined on film. *B*, Spot film showing anterior displacement of barium-filled esophagus by tumor mass.

The patient's postoperative course was uneventful. On the twenty-third postoperative day he vomited approximately 300 c.c. of blood. This was attributed to the presence of a duodenal ulcer. At the time of his discharge from the hospital he was asymptomatic, although roentgenologic studies showed a persistent doudenal deformity. Three months following his discharge he had no complaints referable to either his gastrointestinal tract or thorax.

DISCUSSION

Tortuosity of the descending thoracic aorta producing deformity of the roentgen picture generally is believed to be produced by elongation of the adult aorta between its points of fixation at the heart and diaphragm. This picture is

usually found in elderly individuals with hypertension and arteriosclerosis, although Khoo[2] has reported a case in a 12-year-old boy with a blood pressure of 110 mm. of mercury systolic and 60 mm. of mercury diastolic.

Rosler and White[3] have shown that tortuosity of the thoracic aorta produces deformity of the esophagus in the anterior-posterior plane, as did the case reported here. These authors give a particularly good description of the roentgenologic findings in cases with distorted thoracic aortae.

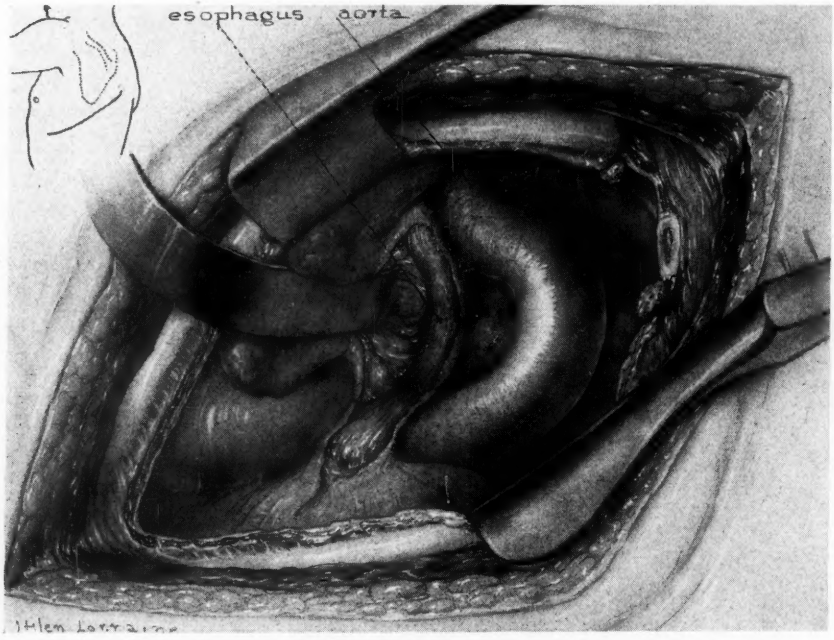

Fig. 3.—Reproduction of anomaly of aorta as seen at operation.

It seems unlikely that elongation in adult life sufficient to produce the extreme tortuosity found in this case could occur in the relatively short segment of aorta lying between the heart and the diaphragm. Assuming that this tortuosity was produced after the position of the heart and diaphragm had become fixed, it would necessitate almost a doubling of the length of the thoracic aorta. A congenital origin would seem more plausible. In early embryonic life the heart lies on the ventral aspect of the pharynx. During the later development of the neck, there is a gradual caudal migration of the heart into the thorax, which produces alterations in the ventral aorta and in the primitive aortic arches. On the right side, the fourth arch descends to the root of the neck, while on the left it is drawn within the thorax. It seems possible that alterations in this mechanism could produce tortuosity of any degree in the descending aorta. This

would also explain the presence of tortuosity unaccompanied by either hypertension or arteriosclerosis in the case reported by Khoo.[2]

A second possible origin of the anomaly herein reported may be found in a study of the fusion of the paired dorsal aortae during early embryonic life. Congdon[4] has shown there is no duplication of the ventral aorta in man, but it is generally agreed that paired dorsal aortae exist. In embryos of about 3 mm. possessing twenty somites, there are two aortic arches and the dorsal aortae are beginning to fuse into a single descending aorta. This fusion takes place by the formation of medial communicating vessels between the two main trunks. These communications gradually coalesce, forming a single tube. If, during this process, a segment of the right or left aorta were obliterated between two communicating branches, the result would be a horseshoe-shaped curve similar to the one encountered in this case.

SUMMARY

A case of an anomaly of the thoracic aorta simulating mediastinal tumor has been reported. Two possible embryologic origins for this condition have been suggested.

REFERENCES

1. Blades, Brian: Mediastinal Tumors. Report of Cases Treated at Army Thoracic Surgery Centers in the U. S., **Ann. Surg. 123:** 749-65, 1946.
2. Khoo, F. Y.: Juvenile Elongation of the Aorta, Am. Heart J. **25:** 404-406, 1943.
3. Rosler, H., and White, P.: Unusual Variations of the Roentgen Shadow of the Elongated Thoracic Aorta, **Am. Heart J. 6:** 768-77, 1931.
4. Congdon, E. D.: Transformation of the Aortic Arch System During the Development of the Human Embryo. Contributions to Embryology of the Carnegie Institute, Washington **68:** 49, 1922.

NERVE-BLOCKING THERAPY FOR FRACTURED RIBS

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THE application of regional nerve blocking to the therapy of disease or injury has most often been made with the primary purpose of controlling the associated pain. This was the object in utilizing paravertebral injections of analgesic solutions to the segmental nerves of fractured ribs as set forth in a preliminary report from this clinic.[1] A limited experience with thirteen patients at that time indicated that such a procedure was definitely useful in controlling pain. It was suggested also that the therapeutic value of such nerve blocking was considerable. Further experiences with 217 patients have served to substantiate the original contention that paravertebral injections of analgesic solutions are indicated to eliminate the pain of fractured ribs and are definitely useful to prevent more serious complications and reduce mortality.

Uncomplicated fractured ribs have little indication for nerve-block therapy other than to relieve pain. The patients are usually ambulatory and have simple therapeutic measures applied, such as adhesive strapping to maintain proper alignment of bone fragments and diminish movements of the involved chest wall. The associated pain is the most annoying complication during healing of the fracture. Such pain cannot be completely controlled by curtailing motion with strapping. Frequently the pain is not felt with respiratory movement only but is persistent or of the type often referred to as callus pain. Infrequently an intercostal neuralgia may accompany rib fracture. Regardless of the type of pain, its relief wins the patient's appreciation and makes it possible to apply desirable fixation of broken fragments without great annoyance. Complete pain relief can be obtained for the great majority of these patients by paravertebral nerve blocking, and the procedure can be depended upon to decrease pain regularly. This report does not substantiate this latter statement with results from extensive clinical experience. It has not been possible to follow accurately the outpatient group treated and the series is not large. However, the results with hospitalized patients with complicated rib fractures indicate that nerve blocking is desirable therapy for the pain of uncomplicated rib fractures.

The unfavorable sequence of chest pain, voluntary splinting of the thoracic wall, suppression of cough, and depression of pulmonary ventilation attendant upon the use of opiates in doses adequate for the relief of pain may predispose to the development of atelectasis, pneumonia, and even death, particularly in cases with extensive injury. According to Berry:[2] "It is generally recognized that contusions of the chest wall may cause a localized atelectasis of the underlying lung with bronchitis or pneumonia, or may activate a quiescent tuberculosis." He states further, "that the best prevention against atelectasis and pneumonia is sufficient sedation to allay pain on respiration and thus prevent too

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much splinting of the injured chest, but yet not enough to depress respiration." There can be little disagreement with the thesis proposed, but it is extremely difficult to maintain the precision balance between sedation which will just relieve pain and the depression of respiration and cough reflex which all too frequently ensue.

It cannot be denied that the effects of opiate administration are not associated with increased pulmonary ventilation but rather the reverse.[3] On the other hand, Harmon, Baker, and Kornegay[4] showed a marked increase in the vital capacity following procaine infiltration into the fracture sites, a phenomenon associated with the obtundation of pain.

In actual practice, the crux of the therapeutic problem involved in the pain of fractured ribs is analgesia which promotes tracheobronchial drainage and normal or increased pulmonary ventilation. Interruption of the painful stimuli by nerve block provides therapy for pain which, unlike the opiates or immobilization, assists in the restoration of normal function in the respiratory tract. Infiltration of the intercostal nerves with one of the local anesthetic agents enables the patient to cough freely without pain, permits the abandonment of voluntary splinting of respiratory movements and possibly releases the bronchial and bronchiolar spasm associated with the chest wall injury.[5] The need for rigid immobilization with plaster or adhesive strapping is eliminated. A fairly loose dressing which does not impede breathing may be useful for fixation in those cases where multiple anterior and posterior fractures have produced a "flail" chest with paradoxical motion.[6] However, it must be emphasized that regional analgesia for fractured ribs does not avoid the necessity of surgical intervention in those patients with severe intrapleural and thoracic visceral injury. It must be considered as a valuable adjunct in those instances. In patients upon whom operative interference is unnecessary, nerve block may be considered the primary therapeutic implement.

Intercostal nerve block has been recommended in the treatment of fractured ribs. Grieco[7] described a group of patients treated by alcohol injections of the intercostal nerves who had quick relief of pain and for whom the period of disability was greatly shortened. Physiologic section of an intercostal nerve with procaine solution is not a difficult procedure if comparatively large amounts of solutions are used, since the infiltration of tissues covers a wide area. It is considerably more difficult to inject alcohol in close contact with an intercostal nerve and, with the small amounts that should be used, denervation may be ineffective except at or very close to the point of injection. If large amounts of alcohol are employed, tissue destruction may be extensive and a complicating neuritis will be more frequent. Moreover, the injection of a single intercostal nerve does not always afford complete relief, since the innervation of the rib by accessory filaments from contiguous nerves is not interrupted. Paravertebral injections are to be preferred and it is recommended that in addition to the nerve corresponding to the fractured bone the next above and below be injected. The technique allows convenient treatment of all fractured ribs that are not complicated by injury to the vertebra or superficial injury at the site of skin puncture.

The technique for paravertebral injection of thoracic segmental nerves is essentially that of Labat[8] and has been described in detail in the original report by Rovenstine and Byrd.[1] The technique for injecting by the intercostal route is a simple one. It is performed by raising a skin wheal along the midaxillary line in the intercostal space below the rib involved. Through this wheal a 5 cm. needle unattached to the syringe is introduced and passed under the lower border of the rib in the direction of the intercostal nerve, and injection is completed with 3 to 5 c.c. of solution. Care must be taken to test for entrance into the pleural cavity by placing a drop of the solution over the opening in the needle. If the drop is sucked in, the pleural cavity has been entered and the needle must be withdrawn and re-inserted. Meticulous aspiration is mandatory to avoid intravascular injection. It is necessary in both methods to block the nerves immediately above and below the injured ribs to secure adequate anesthesia in the fracture site.

Infiltration directly at the site of fracture has been used to some extent but it has several disadvantages. The skin area immediately over the fracture may be contused and unfavorable for the introduction of a needle. Only small amounts of solution can be injected to bathe the involved nerve. The relief of pain with both local infiltration and nerve block by the intercostal route is less complete than following a paravertebral block.

The dangers consequent to the procedures discussed have been pointed out frequently and are attendant upon the possibility of entering the lung, the pleural cavity, a blood vessel, or the subarachnoid space. These accidents are uncommon, are easily recognized, and, therefore, should not produce serious complications. Careful aspiration will obviate injection into the lung tissue or a blood vessel because in both instances blood will be drawn back into the syringe. In addition the injection of procaine into the lung often causes coughing and regularly a metallic taste is noted by the patient. A further precautionary measure is slow injection at the start and, if coughing is induced, immediate cessation and reinsertion of the needle. In paravertebral blocks there is the possibility of injecting into the subarachnoid space either because of an abnormal prolongation of the dura along the nerve beyond the intervertebral foramen or by the introduction of the needle through the intervertebral foramen itself. In either case careful aspiration will demonstrate the presence of cerebrospinal fluid and reinsertion of the needle is indicated.

This report is based upon detailed examination of the results obtained in 217 patients with fractured ribs treated with nerve block at Bellevue Hospital over a period of seven years. This number does not represent the total number of fractured rib cases seen here during this period, but includes only those in whom pain was an important consideration. Some were recent fractures, others old with persistent pain. Many were complicated with other injuries and almost all had more than one rib involved. One hundred and ninety-nine patients were treated with paravertebral somatic blocks, ten with intercostal blocks and eight were given both (Table I).

Only one injection was required in more than 75 per cent of the cases to obtain relief of pain. This figure agrees well with the results of Harmon and as-

sociates[4] who report 25 per cent of their uncomplicated fractures required a second block. In the group receiving paravertebral blocks, 158 were blocked but once, thirty-six were blocked twice, four three times, and one required four injections. Of the patients receiving intercostal blocks, eight required but one block and two were repeated the second time. All the patients in whom both paravertebral and intercostal blocks were done required more than one injection. In all, a total of 280 blocks were completed (Table I).

TABLE I

TECHNIQUE	PARAVERTEBRAL SOMATIC	INTERCOSTAL	COMBINED	TOTAL
Case Incidence	199—91.7%	10—4.6%	8—3.7%	217
Frequency of Block				
One	158—79.4%	8—80%	0	
More than one	41—20.6%	2—20%	8—100%	
Number of Blocks	246—87.8%	12—4.3%	22—7.9%	280
Anesthetic Solutions				
Procaine	64—32.2%	4—40%	1—12.5%	69—31.8%
Procaine and alcohol	119—59.8%	4—40%	2—25%	125—57.6%
Others	16—8.0%	2—20%	5—62.5%	18—10.6%

A variety of solutions were used in the procedure. The most common was procaine (1 to 2 per cent), which in about two-thirds of the cases was followed by the injection of 1 to 2 c.c. of absolute alcohol (Table I). The other solutions used were procaine and ammonium chloride, intracaine, monocaine, and several experimental drugs. The results varied little. When procaine and alcohol was used, there was a slightly longer period of relief from pain than when procaine alone was injected. A single case of alcoholic neuropathy was noted, and there was one instance of painful regeneration of a nerve three months after alcohol block.

Fourteen of the patients had a single rib fractured. The preponderant majority (93.5 per cent) had from two to nine ribs involved and a third of the total number had other complications. Some of these were fractures of other bones, pneumonia, atelectasis, subcutaneous emphysema, hemothorax, pneumothorax, pleural effusion, and trauma of the abdominal viscera (Table II).

TABLE II

COMPLICATIONS PRESENT	NO. PATIENTS
Other Fractures	28
Pneumothorax	8
Pneumonia	16
Subcutaneous Emphysema	12
Atelectasis	2
Pleural Effusion	8
Lacerated Lung	3
Kidney Trauma	2
Chronic Bronchitis	2
Tuberculosis	3
Hemoptysis	3
Head Injuries	10
Laceration of Liver	1
Ruptured Spleen	1

The results in this series were graded as (A) good, providing marked pain relief and permitting the patient to cough freely; (B) moderate, pain relief incomplete but of appreciated value; and (C) poor, no relief obtained. A final classification of "questionable" embraces those cases in which there was a language barrier, an uncooperative patient, or insufficient data recorded. Eighty and six-tenths per cent were classed as good results, 11.9 per cent as moderate, 2.3 per cent as poor and 4.6 per cent as questionable (Table III).

TABLE III

		DEATHS		MORTALITY RATE
		RELATED TO RIB FRACTURES	OTHER CAUSES	
Uncomplicated Cases	142	—	—	—
Good	83.1%			
Moderate	11.3%			
Poor	0.7%			
Questionable	4.9%			
Complicated Cases	75	5	4	12%
Good	76.0%			
Moderate	13.3%			
Poor	6.7%			
Questionable	4.0%			
Total No. Cases	217	Gross Mortality		4.1%
Single Rib Fractured	14			
Multiple Ribs Fractured	203			

There was one death in the series which might be attributed to the procedure. This was a man who was admitted with fractured ribs (left, one to six), hemopneumothorax with paradoxical respirations, a head injury of undetermined extent, and shock. He had been treated successfully for shock for fourteen hours prior to the block. A paravertebral somatic block was completed on the left side (T1-7) with 1½ per cent procaine using a total of 30 c.c. Relief of pain was immediate and skin anesthesia on the left thoracic wall was limited to the area of the nerves blocked. He was sensitive to pain on the contralateral side and below the area blocked on the same side. Six or seven minutes after the block was completed, fibrillary twitchings occurred around his mouth and respirations suddenly ceased. His pulse was unobtainable and he became cyanotic. Endotracheal resuscitation was unsuccessful. Postmortem examination revealed multiple fractures of the ribs and a dilated right heart.

There were eight other deaths in the series, making the gross mortality rate 4.1 per cent (Table III). Three of the deaths were due primarily to pneumonia, one to pneumonia with laceration and contusion of the liver, one to pneumonia and subcutaneous emphysema of the entire body, and one to pneumonia and laceration of the brain with intracranial hemorrhage. One autopsy revealed an extensive pneumothorax with laceration of the lung. The eighth death in the series occurred in a woman who developed delirium tremens and died two days later. The mortality rate in this series is less than in the series of fractured ribs reported by Knoepp,[9] none of whose patients were treated by nerve block. In his series, all patients with uncomplicated cases survived but, when complications were present, mortality varied from 23 to 71 per cent depending

upon the severity of the complications. In this series none of the uncomplicated cases ended in death but of the complicated ones only 12 per cent terminated in death.

In the majority of instances the relief of pain was immediate following the block and the patient was able to cough freely. Some of these had radiographic evidence of pneumonia processes with elevation of temperature, and within a day or two after a successful block remarkable clearing was demonstrated and the temperatures had returned to or near normal. The ability to cough and to ventilate more fully is responsible undoubtedly for such satisfactory recovery.

In addition to the patients with fractured ribs reported here, paravertebral and intercostal blocks have been used frequently to treat postoperative pain after a variety of thoracic and upper abdominal operations. Regularly, the relief of pain with its attendant freedom to cough and breathe without restraint has brought about considerable clinical improvement, and aided in securing a more normal convalescence. Gius[10] reports similar results in children who developed postoperative atelectasis following appendectomy.

SUMMARY

A survey of 217 patients with fractured ribs treated with nerve blocking, either paravertebral or intercostal, is presented. For more than 80 per cent of these, there was almost immediate relief of pain following the initial injection. Less than 25 per cent required more than one treatment. Among the patients presented, 93.5 per cent had multiple rib fractures and more than a third of the entire series had major complications associated with trauma. The mortality rate in the complicated cases was 12 per cent and the total mortality was 4.1 per cent. These results indicate that pain relief with nerve block is of significant value in preventing pulmonary infections and death of patients with fractured ribs.

This report was prepared under the supervision of E. A. Rovenstine, M.D.

REFERENCES

1. Rovenstine, E. A., and Byrd, M. L.: Use of Regional Nerve Block During Treatment for Fractured Ribs, Am. J. Surg. **46**: 303-308, 1939.
2. Berry, F. B.: Wounds of Thoracic Viscera, Am. J. Surg. **39**: 12-17, 1938.
3. Capelle, W.: Die Bedeutung des Wundschmerzes und seiner Ausschaltung für den Ablauf der Atmung bei Laparotomierten, Deutsche Ztschr. f. Chir. **246**: 466-470, 1936.
4. Harmon, P. H., Baker, D. R., and Kornegay, R. D.: Uncomplicated Fractures of Ribs and Major Injuries of Chest Wall, J. A. M. A. **118**: 30-34, 1942.
5. DeTakats, G., Fenn, G. K., and Jenkinson, E. L.: Reflex Pulmonary Atelectasis, J. A. M. A. **120**: 686-690, 1942.
6. Samson, P. C., and Fitzpatrick, L. J.: Intercostal Nerve Block; Its Role in Management of Thoracic Casualties, Calif. & West. Med. **62**: 254-256, 1945.
7. Grieco, Fausto: Alcohol Injection of Intercostal Nerves in the Treatment of Fractured Ribs, Riv. de Chir. **3**: 507-515, 1937.
8. Labat, G.: Regional Anesthesia, ed. 2, Philadelphia, 1928, W. B. Saunders Company.
9. Knoepp, L. F.: Review of 386 Cases of Fractured Ribs, Am. J. Surg. **52**: 405-414, 1941.
10. Gius, J. A.: Paravertebral Procaine Block in Treatment of Postoperative Atelectases; Preliminary Report, Surgery **8**: 832-838, 1940.

CONTINUOUS SPINAL ANESTHESIA FOR THORACOPLASTY

A Report of 300 Operations

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THE introduction of continuous spinal anesthesia provided the surgical profession with new opportunities. Once this method was proved safe and free from complications, the possibilities were evident that procedures of greater magnitude could be carried out, since the duration of anesthesia could be maintained safely as long as necessary. In 1941, Lemmon[1] reported a series of 500 operations performed under continuous spinal anesthesia with no anesthetic deaths and no neurologic complications. Numerous reports in the last five years have confirmed the safety of the method.

This report, we believe, is the first to describe a series of major thoracic procedures performed under continuous spinal anesthesia. This series was begun in 1941, after a review of the pharmacology and the physiologic effects of procaine in the cerebrospinal fluid led to the conclusion that sufficiently high levels of anesthesia could be induced and maintained with continuous spinal technique to perform any stage of thoracoplasty, apicolysis, extrapleural pneumothorax, and similar procedures. This paper covers 300 thoracoplasties performed under continuous spinal anesthesia from February, 1941, to December, 1944.

PHARMACOLOGY AND PHYSIOLOGIC EFFECTS OF PROCAINE IN CEREBROSPINAL FLUID

Hatcher and Eggleston,[2] in 1916, demonstrated that the toxicity of Novocaine (procaine) given intravenously, depended largely upon the concentration of the drug and the rate of injection. Comparatively small doses given rapidly in high concentrations were fatal to animals, whereas large doses could be tolerated if given slowly and in low concentrations. They also showed that procaine was destroyed in the liver and not in the kidneys, as formerly supposed.

Bullock and MacDonald[3] showed in experiments on cats that the concentration of procaine in the cerebrospinal fluid falls rapidly from the point of injection where the concentration is highest, to the cisterna, where the concentration is negligible. Increasing the concentration, increased the duration of anesthesia. Only minimal amounts were found to be absorbed into the blood. The average amount found in the blood of human beings under spinal anesthesia was less than 0.002 mg., although as much as 300 mg. had been given intrathecally.

In 1942, Burford,[4] discussing the problem of absorption of procaine from the cerebrospinal fluid, concluded that the drug tended to become fixed to the immediately adjacent neural elements and that any absorption that took place was slow. The probable pathways of absorption are (1) through the spinal arachnoid villi and (2) through direct absorption

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from the capillary bed throughout the parenchyma after passage along the perivascular extensions of the subarachnoid space. He stated that "there is never a significant, still less a dangerous concentration of the drug in the blood."

After introduction into the subarachnoid space, the drug produces a progressive anesthesia from the sensory to the motor level. Experience has shown that small quantities of procaine suffice to maintain the desired depth of anesthesia once the initial block is established, since the greater part of the required dose is utilized in effecting the initial block.

Various explanations have been offered for the cause in the fall of blood pressure often seen during spinal anesthesia. This phenomenon has been attributed to decreased cardiac output, increase in the visceral capillary bed through trauma, handling of the intestinal tract, and to vasomotor paralysis.

Bradshaw,[5] in 1936, showed that the fall in pressure in experimental animals is due to vasomotor paralysis. He recorded the blood pressure of cats after the intrathecal injection of procaine and found the usual fall. After total sympathectomy there was no change in blood pressure after equal doses of spinal procaine. He concluded that the most important fibers concerned in the fall of blood pressure are those below the fifth thoracic segment. Bradshaw also stated that respiratory failure occurs only when high concentrations of procaine reach the region of the cervical cord and that this rarely happens in man except inadvertently. He suggested that the occasional collapse seen may be due to accidental injection of the drug into a dural vein. In this respect, the finding of Hatcher and Eggleston[2] should be remembered; namely, that procaine rapidly injected intravenously is extremely toxic. The problem in fall of blood pressure has been overcome in modern practice by the use of ephedrine or similar drugs prior to the induction of spinal anesthesia and the maintenance of circulating blood volume by the use of intravenous fluids during the course of operation.

A contribution to the problem of controlling spinal anesthesia was made by Koster and associates[6] when they showed that the duration of the paralysis of the lower extremities during spinal anesthesia could be shortened markedly by withdrawing cerebrospinal fluid. This observation, plus our own experience and that of others, shows that fixation of the drug in the neural elements can be but of short duration and that some other explanation must be given before we can understand anesthesia produced by procaine.

The fact that effective levels of anesthesia can be reached using much lower concentrations of procaine than originally recommended has definitely increased the safety of the anesthetic. We have progressively reduced the concentration employed to our present routine use of 2½ per cent solution.

In summary, the problems to be overcome in the use of (thoracic) spinal anesthesias were (1) fall in blood pressure, (2) toxicity of the agent employed, and (3) lack of control over the injected drug. These have been met by (1) the use of ephedrine and fluids to prevent and overcome the fall in blood pressure, (2) the use of procaine as a known safe agent, and (3) the use of continuous spinal as a method of controlling the level of anesthesia effectively for long periods of time.

CONTINUOUS SPINAL ANESTHESIA IN THORACIC SURGERY

It must be emphasized that spinal anesthesia has little, if any, place in intrapleural surgery. Here the anesthetic method of choice is inhalation anesthesia given through an endotracheal tube so that respirations can be controlled and bronchial secretions can be removed. However, in operations upon the chest wall, such as thoracoplasty, we believe continuous spinal anesthesia has advantages no other anesthetic offers.

The first patient in this series was operated on at Eagleville Sanatorium in February, 1941. The first patient at Jefferson Hospital was operated on by Dr. H. H. Bradshaw in March, 1941. From February, 1941, to December, 1944, 300 thoracoplasties were attempted under continuous spinal anesthesia. These

operations were done on 114 patients, ages ranging from 19 to 59. For purpose of study, we have divided the cases into two groups (1) unilateral pulmonary tuberculosis and (2) bilateral pulmonary tuberculosis. A number of these patients have had pneumothorax maintained in the contralateral lung while undergoing staged thoracoplasty. Forty-nine patients had a Semb extrafascial apicolysis done in addition to the first stage thoracoplasty, an operation which adds substantially to the magnitude of the procedure.

DOSAGE

There is no arbitrary dose which can be advised. *Enough procaine is given to reach and maintain the desired level of anesthesia.* This amount varies greatly from stage to stage in the individual patient. The average initial dose has been 100 mg. of procaine and the total dose has varied from as little as 150 mg. to as much as 1,000 mg. It must be emphasized that the amount required is unpredictable. However, the majority, or 65 per cent of the stages, have been done with a total dosage of procaine ranging from 200 mg. to 400 mg. It should be observed at this point that with the solution now used, 2½ per cent procaine in spinal fluid, there is apparently very little danger of untoward effects on respiration, even when the level reaches higher than is desired. It is known that higher concentrations of procaine are needed to cause motor paralysis than sensory anesthesia. Apparently the dilution of procaine in the spinal fluid is such that it reaches the cervical region in amounts insufficient to cause paralysis of the diaphragm while concentrated enough to cause sensory loss and provide muscular relaxation. We have seen many patients with complete anesthesia of the scalp and tongue with no sign of respiratory difficulty.

EFFECT ON PULSE RATE, BLOOD PRESSURE, AND RESPIRATION

There was no appreciable difference in the effect of spinal procaine on the pulse rate in either the unilateral or bilateral group. Of the unilateral patients, 11⅓ per cent and of the bilateral patients, 15 per cent showed a fall in pulse rate at the conclusion of the operation. A rise in pulse rate averaging twenty-five per minute was shown in 28⅔ per cent of the unilateral and 27⅔ per cent of the bilateral cases. There was no change in pulse rate in 5⅔ per cent of the unilateral and 5⅓ per cent of the bilateral cases. In the remaining 1⅓ per cent of the unilateral and 1⅔ per cent of the bilateral cases, complete records were not kept.

As in pulse rate, there was no significant difference in the change in blood pressure in each group. A fall in blood pressure was shown at the conclusion of the operation in 30⅓ per cent of the unilateral and 35⅓ per cent of the bilateral cases. A rise in blood pressure was noted at the termination of the surgical procedure in 6⅔ per cent of the unilateral and 5⅔ per cent of the bilateral cases. In 2 per cent of the unilateral and 1⅔ per cent of the bilateral cases there was no change in blood pressure. In the remaining 8 per cent of the unilateral and 7 per cent of the bilateral cases the desired information was not recorded.

In 29⅓ per cent of the unilateral and 33⅓ per cent of the bilateral cases there was a slight rise in respiratory rate, averaging four per minute. In ⅔ per cent of each group there was a fall in respiratory rate and in 12⅓ per cent of each group no change in respiratory rate was noted. In the remaining 4⅔ per cent of the unilateral and 3⅓ per cent of the bilateral cases, the necessary data was not recorded.

In ⅓ per cent of the *entire* group of unilateral cases and 3 per cent of the *entire* number of bilateral cases, operation was discontinued, for reasons stated later, as tabulated below. There was one patient with temporary arrest which responded promptly to oxygen, withdrawal of spinal fluid, and artificial respiration. There was one anesthetic death which will be discussed shortly.

TABLE I

	PULSE				BLOOD PRESSURE				RESPIRATIONS				DISCONTINUED OPERATIONS
	FALL	RISE	NO CHANGE	?	FALL	RISE	NO CHANGE	?	FALL	RISE	NO CHANGE	?	
Unilateral													
141 cases (47%)	34	86	17	4	91	20	6	24	2	88	37	14	1
Percentage	11⅓	28⅔	5⅔	1⅓	30⅓	6⅔	2	8	⅔	29⅓	12⅓	4⅔	⅓
Bilateral													
149 cases (49⅔%)	45	83	16	5	106	17	5	21	2	100	37	10	9
Percentage	15	27⅔	5⅓	1⅔	35⅓	5⅔	1⅓	7	⅔	33⅓	12⅓	3⅓	3

SUPPLEMENTARY ANESTHESIA

In 35 patients it was necessary to use one or more types of supplemental anesthesia. The percentage of operations thus requiring supplemental anesthesia was 11.7, the effectiveness of continuous spinal anesthesia in this series being 88.3 per cent. When the 10 cases which were discontinued are added to this group, the total effectiveness of the continuous spinal anesthetic method was 85 per cent.

COMPLICATIONS

Aside from the ten patients on whom operations had to be discontinued before completion, nine having bilateral lesions, there was no appreciable difference in the incidence of complications in the two groups. This is a very difficult subject to discuss and evaluate since we are attempting to separate the complications of the anesthetic from those inherent in the operative procedure itself. In many cases the differentiation cannot be made.

Of the operations, 4⅓ per cent were followed by nausea and 13 per cent by vomiting. This compares very favorably with the incidence of these phenomena following general anesthesia. While we are not able to furnish the figures as to the incidence in the cases done under cyclopropane and ether, it is our impression that nausea and vomiting to some degree followed the great majority of cases.

Postoperative headaches occurred in 9.3 per cent of cases. In none of these was the headache alarming or persistent. The percentage of headaches found

in our cases was larger than in most reported series of infradiaphragmatic procedures done under continuous spinal anesthesia. However, we feel that this minor complication is a small price to pay for the many compensating advantages of the method.

Two patients, or 0.66 per cent went into a state of shock or collapse following continuous spinal, but responded to the usual methods of resuscitation. Whether this can be attributed to the anesthetic method is very questionable, since a far greater number was seen when inhalation anesthetics were being used.

In ten cases, it was necessary to discontinue the operation because of difficulties with the anesthetic or unsatisfactory condition of the patient. Although it is quite likely that some of these patients would have presented the same difficulties under inhalation anesthesia we are listing them as failures of the method. The following are brief summaries of the ten discontinued cases:

Case 1. Unilateral pulmonary tuberculosis. Anesthetic unsatisfactory since free flow of spinal fluid could not be obtained.

Case 2. Bilateral pulmonary tuberculosis. Free flow of spinal fluid obtained only after third puncture. Fluid was bloody. Satisfactory level was not obtained after administration of 500 mg. of procaine.

Case 3. Bilateral pulmonary tuberculosis. Satisfactory level was not obtained after 500 mg. of procaine had been given.

Case 4. Bilateral pulmonary tuberculosis. Patient had respiratory arrest during course of operation. Prompt recovery with oxygen, artificial respiration, and withdrawal of spinal fluid.

Case 5. Bilateral pulmonary tuberculosis. Marked cyanosis developed and operation was discontinued.

Case 6. Bilateral pulmonary tuberculosis. Patient had alarming rise in pulse rate and marked venous oozing. Extreme indrawing of intercostal spaces present after exposure of costal cage. Similar experience with this patient later, using regional block anesthesia with 1 per cent procaine solution.

Case 7. Bilateral pulmonary tuberculosis. Anesthesia could not be obtained above level of third thoracic vertebra.

Case 8. Bilateral pulmonary tuberculosis. Patient's blood pressure fell to 60/40 following skin incision and mobilization of scapula.

Case 9. Bilateral pulmonary tuberculosis. Anesthetic level satisfactory but patient was extremely restless and uncooperative. This case may represent one of untoward effects of premedication with barbiturates.

Case 10. Bilateral pulmonary tuberculosis. Anesthetic level satisfactory but patient restless and apprehensive. Very similar to Case 9.

MORTALITY

There was one anesthetic death in this series of 300 operations. This occurred early in our experience, in 1941. This patient was a white male, aged 37. Examination revealed bilateral pulmonary tuberculosis with extensive disease in the left lung and old fibrotic lesions in the right upper lobe. Sputum averaged 35 c.c. daily and was highly positive for tubercle bacilli. First stage thoracoplasty was carried out uneventfully on Nov. 10, 1941. On December 19, patient was scheduled for a second stage thoracoplasty. The spinal tap was bloody and a free flow was not obtained. These two contraindications to spinal anesthesia were ignored by the surgical team of the day and the patient was given 150 mg.

of procaine in a 5 per cent solution. Barbotage was used to extend the anesthetic solution upward. Two subsequent doses of procaine were given in 150 mg. and 100 mg. doses, disregarding the usual dosage of 40 to 60 mg. at two-minute intervals.

Oxygen was not given before cyanosis and restlessness were observed.

One hour after induction of the anesthesia and almost immediately after the last dose of 100 mg. of procaine was given, respiratory arrest occurred, followed momentarily by cardiac arrest.

Spinal fluid could not be withdrawn until a puncture at another level was made.

Artificial respiration with 100 per cent oxygen administration brought no response.

We believe that had the rules for the anesthetic been adhered to this fatality would not have occurred.

TECHNIQUE

Enough barbiturate is given the evening before operation to ensure a restful night. Two hours prior to operation 1½ grains of Nembutal or Seconal is given, and one hour prior to operation the patient is given ¼ grain of morphine sulfate and 1/150 grain of scopolamine, hypodermically. The patient is brought to the operating room with as little disturbance as possible. Many patients are drowsy but can be aroused easily. The patient is placed on the table in the position to be maintained during operation, the lateral decubitus. Ephedrine sulfate, ⅜ grain, is given hypodermically and a skin wheal is raised in the lumbar region over the widest interspace. Choice of interspace is unimportant. A small skin incision is made and the introducer is inserted through the interspinous ligament. A No. 19 gauge malleable needle is used. If bloody fluid is obtained, the operation is postponed. Upon entering the subarachnoid space the spinal fluid pressure is taken, after which a syringe is connected to the needle and it is determined whether or not a free flow of spinal fluid is present. This is highly important, since safe continuous spinal anesthesia for thoracic procedures cannot be carried out if a free flow of fluid is not present. Should the flow be unsatisfactory, a second puncture is made at another interspace.

When a free flow of fluid has been obtained, the malleable needle is bent so that it is parallel to the skin and then fixed with adhesive tape. A 30-inch rubber tube, with a volume capacity of 2 c.c. is then attached to the needle. Spinal fluid, 20 c.c., is then drawn into a syringe containing 500 mg. of procaine.

The initial dose is usually 100 mg. The table is then placed in a 15° Trendelenburg position. Subsequent 50 mg. doses of procaine are given at two-minute intervals until the desired level of anesthesia is obtained. The rate of ascent is usually two or three interspaces with each 50 mg. dose. It is rarely necessary in a first stage thoracoplasty to give more procaine after the level has reached the upper border of the scapula. While the patient is prepared and draped, the anesthesia level will rise to the angle of the jaw, the desired point to which anesthesia must rise for a first stage procedure and must be maintained at this level until closure of the upper angle of the incision has progressed to the midscapular area. At this stage of the operation the stopcock on the tubing is opened and spinal fluid is allowed to flow into the syringe. The amount of fluid removed varies with each patient and is dependent on the degree of sensation and motor function regained.

Removal of spinal fluid is discontinued if abdominal respiratory movements are present, if the patient can move the toes, or if the sensory level is in the midscapular area, at the conclusion of the operation.

The needle is not removed from the subarachnoid space until this degree of motor function or sensation has been restored.

Occurrence of nausea and retching is very uncommon since we discontinued barbotage and a 5 per cent solution, in 1942. If these symptoms do occur it is during induction or early in the course of the operation and are promptly relieved by withdrawal of spinal fluid; usually 2 to 4 c.c. will suffice. After a two- or three-minute interval, small doses of the drug are injected as needed.

Oxygen by mask is given to all patients as soon as the operation is begun.

ADVANTAGES

During an operation under continuous spinal anesthesia, there is excellent muscular relaxation. Mobilization and retraction of the scapula is facilitated. Respirations are quiet and not labored and paradoxical motion of the lung is minimized. Cyanosis is unusual and the shock so often seen when the patient is moved from the operating table after inhalation anesthesia is almost nonexistent.

These patients require much less nursing care than those operated upon under inhalation anesthesia. Recovery is prompt and uneventful. Secretions in the bronchial tree are much less likely to be aspirated into the contralateral lung since at no time after operation is there loss of the cough reflex.

Patients in both hospitals requiring thoracoplasty prefer the continuous spinal method of anesthesia. The patients who have had previous stages done under inhalation anesthesia and subsequent ones under spinal anesthesia are unanimous in their preference for spinal anesthesia because of the comfort during operation and the prompt, uneventful recovery.

CONCLUSIONS

1. Continuous spinal is a safe anesthetic for extrapleural thoracic procedures.

2. The operative procedure is made easier because of quieter breathing, better muscular relaxation, and lessened paradoxical motion of the lung.

3. Recovery is prompt, shock is rare, and postoperative nursing requirements are reduced to a minimum.

4. Postoperative complications are fewer than those occurring after inhalation anesthesia for similar operative procedures.

REFERENCES

1. Lemmon, W. T., and Pascal, Jr., G. W.: Continuous Spinal Anesthesia, Pa. M. J. **44**: 975, 1941.
2. Hatcher, R. A., and Eggleston, E.: A Contribution to the Pharmacology of Novocain, J. Pharmacol. & Exper. Therap. **8**: 385, 1916.
3. Bullock, K., and MacDonald, A. D.: The Fate of Drugs Used in Spinal Anesthesia, J. Pharmacol. & Exper. Therap. **62**: 39, 1938.
4. Burford, G. E.: The Tolerance of Humans for Procaine Injected into the Subarachnoid Space, Anesthesiology **3**: 159, 1942.
5. Bradshaw, H. H.: The Fall in Blood Pressure During Spinal Anesthesia, Ann. Surg. **104**: 41, 1936.
6. Koster, H., Shapiro, A., Warshaw, R., and Margolick, M.: Removal of Procaine from the Cerebrospinal Fluid During Anesthesia, Arch. Surg. **39**: 682, 1939.